notespaedia

PEDIATRICS IMAGE BANK

DESIGNED BASED ON LAST 3 YEARS EXAM PATTERN

PAEDIATRICS

Image Bank

We hope you enjoy using these notes

They have been hand-crafted with an obsessive attention to details, with aim of capturing the course's content in a way that's right for you.

Thank You

For the Students
from the Students

notespaedia

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HOW TO USE THIS NOTE

Image Bank Series

- High Quality Images
- Includes last three years NEET PG, AIIMS and new pattern INICET Image based questions
- Probable images that can be tested in future exams based on predictability and weightage are included.
- Referred from the latest Standard textbook.
- Direct reference from National faculty lectures.

CONGRATULATIONS

You now posses the book that will guide Medical PG Aspirants to score the best rank in PGMEE. With appropriate care, the binding should last the useful life of the book. Keep in mind that putting excessive flattening pressure on any binding will accelerate its failure. If you purchased a book that you believe is defective, please **immediately** return it to the place of purchase.

FOR BETTER RESULTS

Use as revision book :

Use this notes for revision as a supplement to your primary resource. These notes are specifically designed for active recall and visual memory for reinforcing the revision strategy. Best results are gained on quick recap a week before exam.

Consider *IMAGE BANK* as your annotation hub :

Annotate material from your primary resource, such as lecture class or videos, into your notes. We have already referred and incorporated almost all information from the available sources. This will keep all the high-yield information you need in one place.

BOX ANNOTATION :

We have introduced concept of Box annotation

1. **RED BOX :** Includes the most repeated topic along with the next potential MCQ that we believe will be tested in future exam
2. **BLUE BOX :** Includes Previous AIIMS/ NEET PG /INICET recalls

COLOR CODE:

The notes are specifically color coded as follows

1. **BLACK :** Definition
2. **RED :** Clinical features
3. **BLUE:** Pathogenesis / Mechanism of Action
4. **VIOLET:** Investigation, Radiography and Lab Diagnosis
5. **GREEN:** Treatment

Prime Your Memory :

The visual representation of concepts incorporated into the notes serve as a useful way of retaining key associations and keeping high-yield facts fresh in your memory just prior to exams.

CONTRIBUTE TO ELITE NOTES :

No resource is error free and we do not guarantee the notes are free of errors. If you feel you want to add more contents or wish to be a part of Content Dev please contact us at **support@notespaedia.com**

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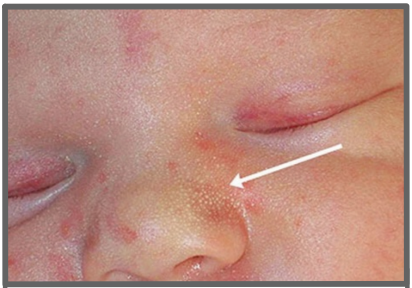
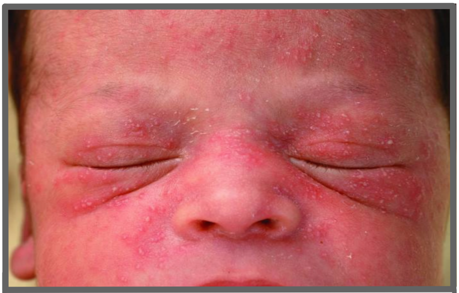
FINDINGS IN NEWBORN



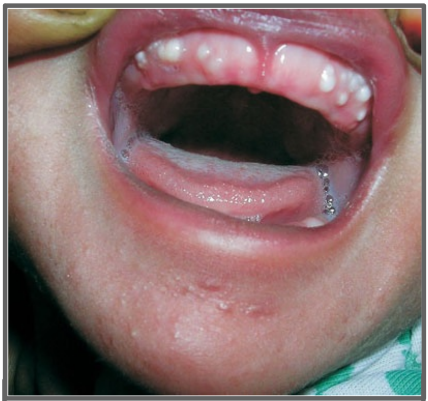
MILIA

- Distended sebaceous glands on face and neck
- Normal skin finding



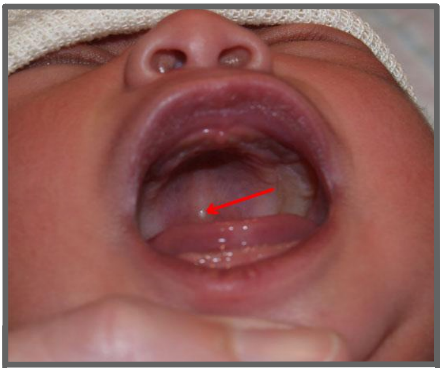
ERYTHEMA TOXICUM

- Most common normal skin finding on 2nd or 3rd day
- Red coloured papules and pustules - eosinophilia infiltrates
- Appears on trunk and face, never at birth



BOHN'S NODULE

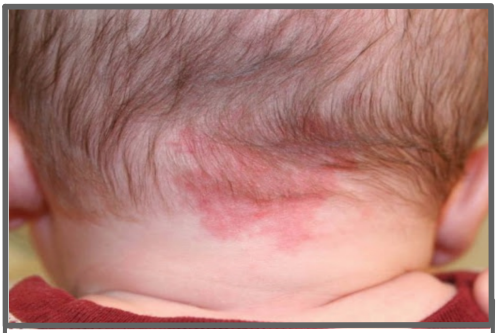
- Epithelial inclusion cyst
- Seen in gingiva



EPSTEIN PEARLS

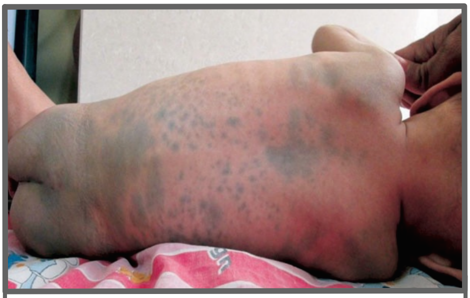
- Epithelial inclusion cyst
- Seen in hard palate

Reference: Essential Pediatrics Ghai (Pg 124)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 217)



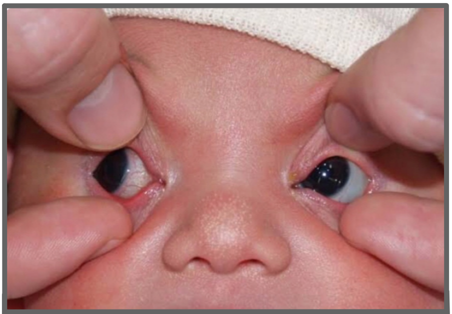
SALMON PATCH/ STROKE BITE

- Commonly seen in Nape of neck and forehead
- Due to dilated blood vessels (vascular ectasias)
- Rx: Regress by 1- 3 yrs

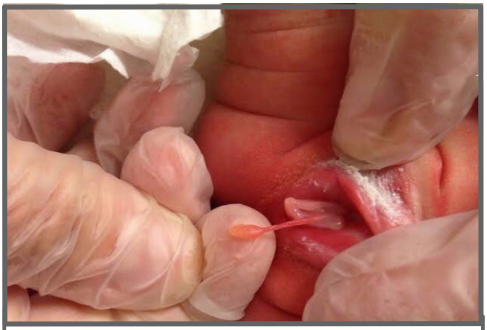


MONGOLIAN SPOT

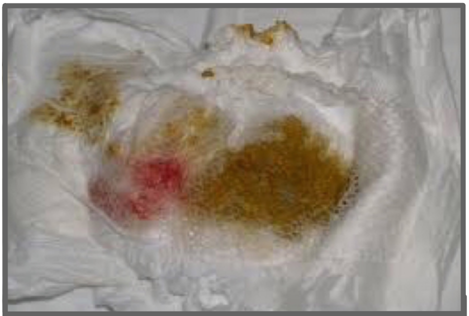
- Congenital dermal melanocytosis
- Macule due to increased melanocytes
- Seen in lumbosacral area
- Disappear by 1 year of age



SUB CONJUNCTIVAL HEMORRHAGE

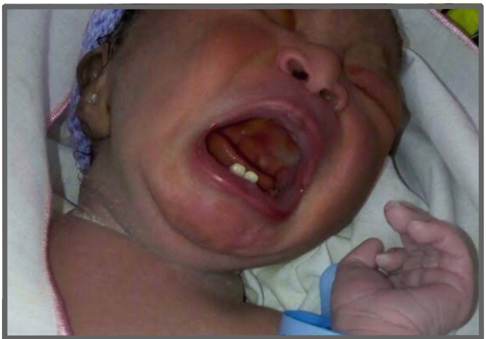


HYMEN TAGS



WITHDRAWAL VAGINAL BLEEDING

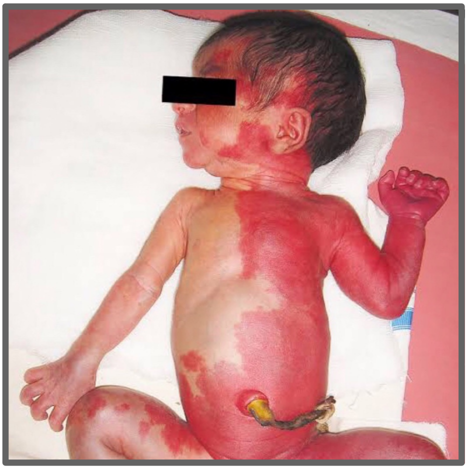
2-5 day of life



NATAL TEETH (PURE DECIDUOUS TEETH)

Lower incisors - most common

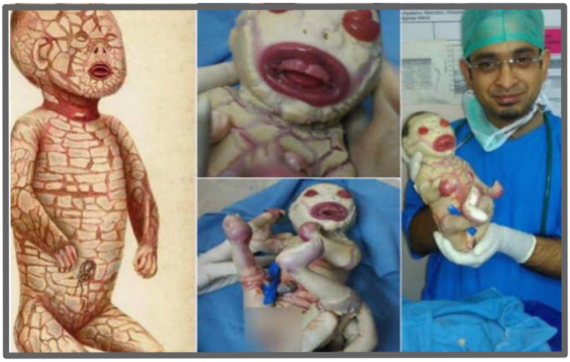
HARLEQUIN COLOUR CHANGE



[redacted]

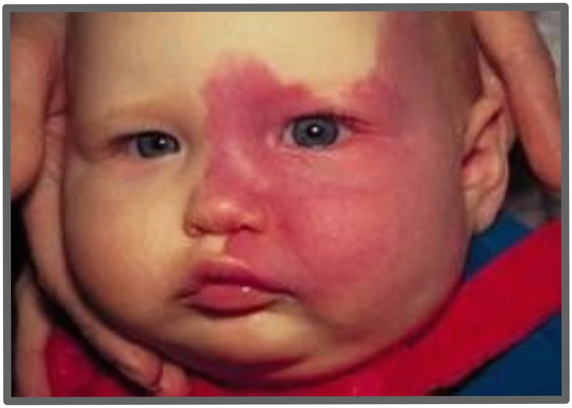
Due to autonomic dysfunction of cutaneous blood vessels

Differential Diagnosis → **HARLEQUIN ICTHYOSIS**

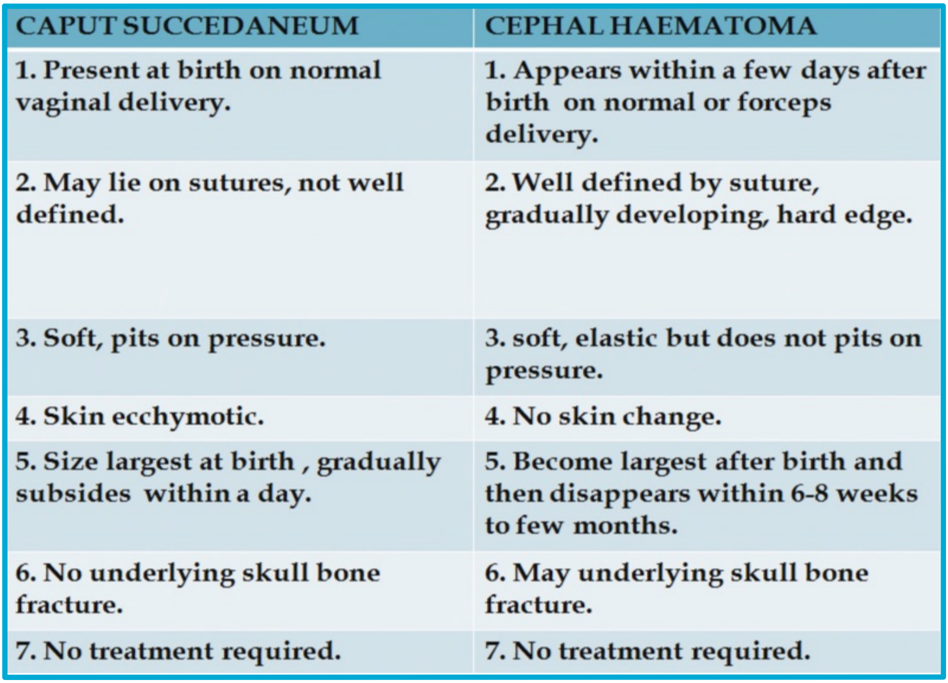


HARLEQUIN ICTHYOSIS
(Not a transient phenomenon)

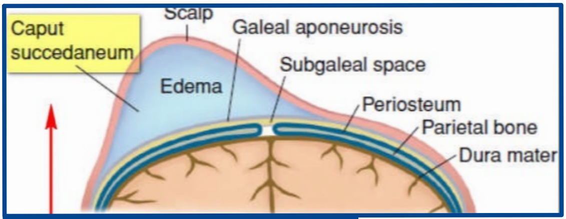
PORT WINE STAIN/ STURGEON WEBER SYNDROME

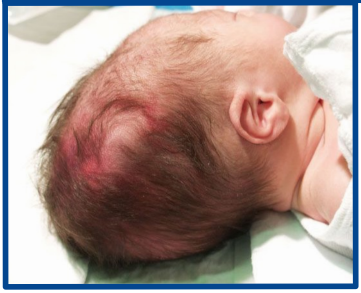


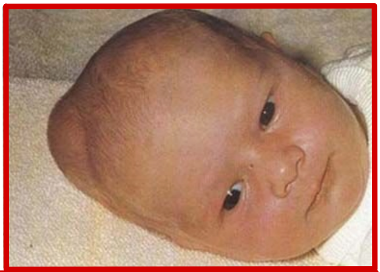
- Sporadic
- Neurocutaneous disorders
- Also known as nevus flammeus
- Appears at birth
- Persist throughout life.

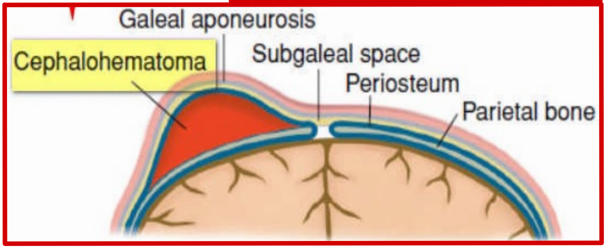


CAPUT SUCCEDANEUM	CEPHAL HAEMATOMA
1. Present at birth on normal vaginal delivery.	1. Appears within a few days after birth on normal or forceps delivery.
2. May lie on sutures, not well defined.	2. Well defined by suture, gradually developing, hard edge.
3. Soft, pits on pressure.	3. soft, elastic but does not pits on pressure.
4. Skin ecchymotic.	4. No skin change.
5. Size largest at birth , gradually subsides within a day.	5. Become largest after birth and then disappears within 6-8 weeks to few months.
6. No underlying skull bone fracture.	6. May underlying skull bone fracture.
7. No treatment required.	7. No treatment required.

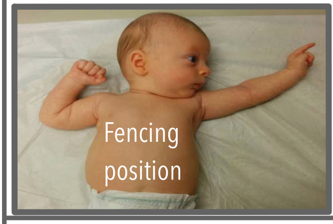
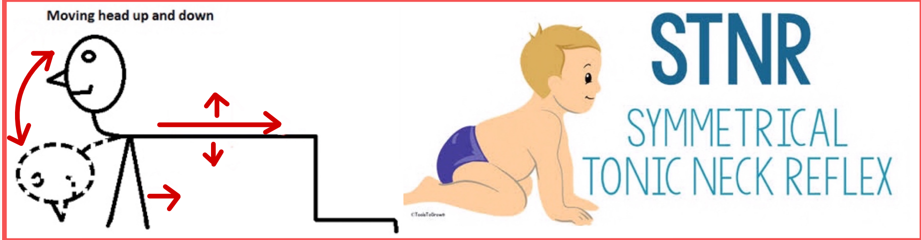
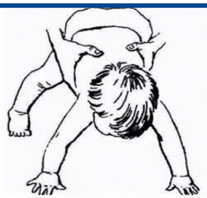
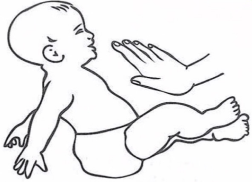
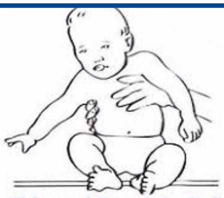

Caput succedaneum
Scalp
Galeal aponeurosis
Subgaleal space
Edema
Periosteum
Parietal bone
Dura mater

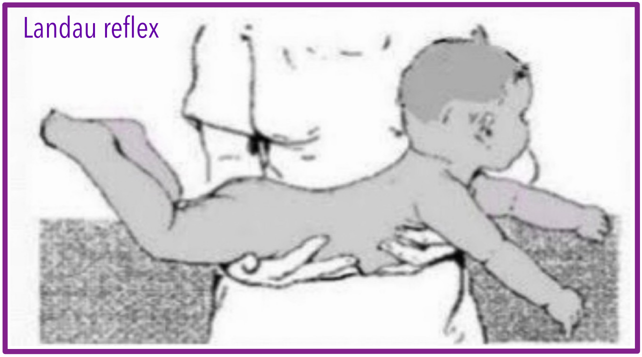




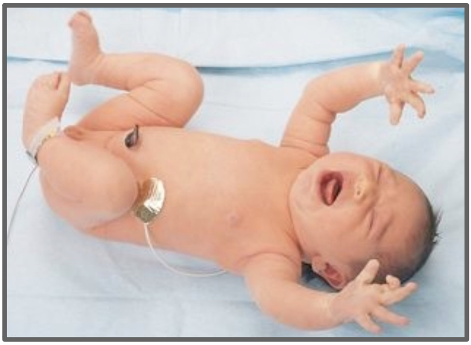

Galeal aponeurosis
Cephalohematoma
Subgaleal space
Periosteum
Parietal bone

NEONATAL REFLEXES

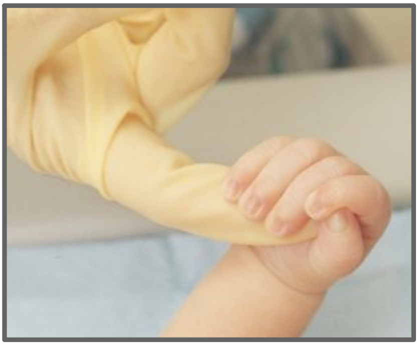
REFLEX	NORMAL TIME OF APPEARANCE	NORMAL TIME OF DISAPPEARANCE
SUCKING REFLEX	22 WEEKS GESTATION	3 MONTHS OF AGE
ROOTING REFLEX	28 - 32 WEEKS GESTATION	3 MONTHS OF AGE
PALMAR GRASP (ulnar palmar grasp Reflex) C8 – T1 Hypothenar region	32 WEEKS (IMMATURE MORO)	3 MONTHS OF AGE
ASYMMENTRIC TONIC NECK REFLEX 	36-37 WEEKS	4-5 MONTHS
MORO'S REFLEX	36- 37 WEEKS (MATURE)	3-6 MONTHS
SYMMENTRIC TONIC NECK REFLEX	1 - 2 MONTHS OF POSTNATAL LIFE	7-8 MONTHS
 Moving head up and down STNR SYMMETRICAL TONIC NECK REFLEX		
PARACHUTE REFLEX	2-4 MONTHS OF POSTNATAL LIFE	PERSIST THROUGHOUT LIFE
 **Forward Parachute Reflex** (Protective Extension Reaction Forward)	 **Backward Parachute Reflex** (Protective Extension Reaction Backward)	 **Sideward Parachute Reflex** (Protective Extension Reaction Sideward)

LANDAU REFLEX Flying superman reflex Done on horizontal suspension ventrally ↓ On extension of neck ↓ Extension of upper and lower limb ↓ On flexion of neck ↓ Flexion of upper and lower limb	**3-4 MONTHS OF POSTNATAL LIFE**	**DISAPPEARS AT 9-10 MONTHS**
 Landau reflex		

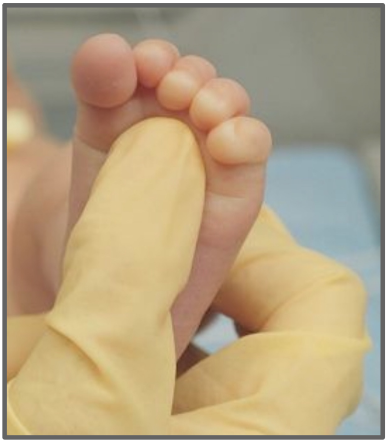
MORO REFLEX



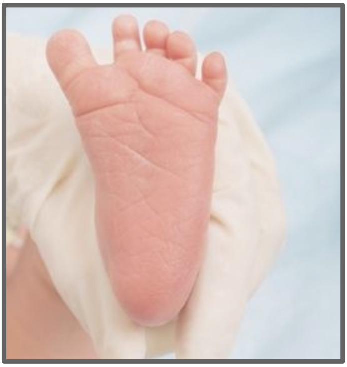
PALMAR GRASP REFLEX



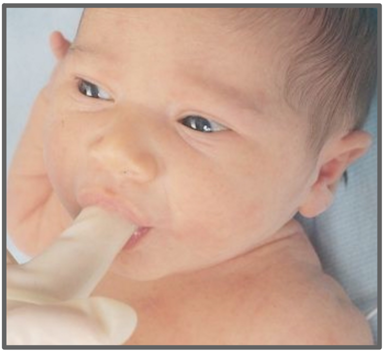
PLANTAR GRASP REFLEX



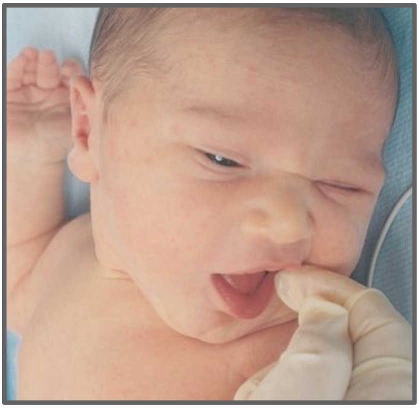
BABINSKI REFLEX



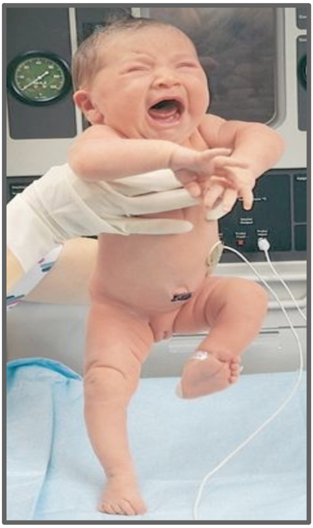
SUCKING REFLEX



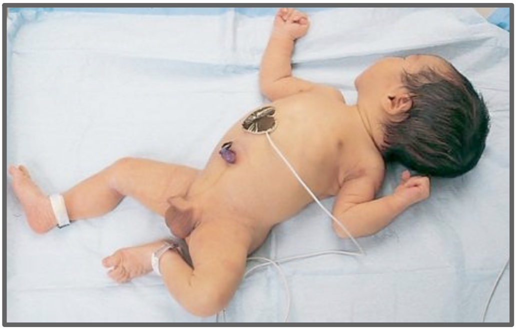
ROOTING REFLEX



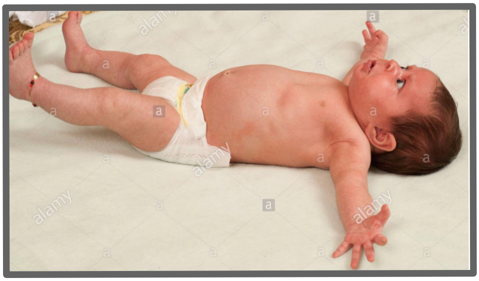
STEPPING REFLEX



TONIC NECK REFLEX



MORO REFLEX



- Fully developed at 37 weeks of gestation
- Duration- 5-6 months
- Absent or incomplete moro reflex is normal in a preterm baby
- Initially extension and abduction then Flexion and Adduction

Absent Moro reflex in term:

- Anencephaly(brain anomalies)
- HIE(sequels of birth asphyxia)- stage 3

Unilateral Moro reflex:

- Erb's palsy
- Fracture clavicle
- Dislocation

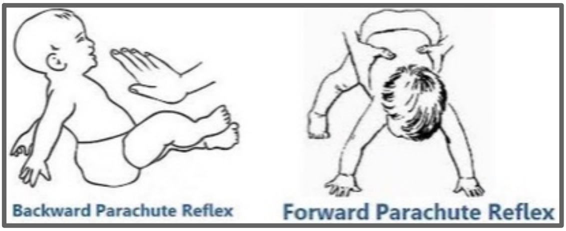
Sluggish Moro reflex: hypotonia

- Down syndrome,
- Severe Asphyxia,
- Spinal muscular atrophy

Persistence of Moro reflex: > 6months

- Development delay eg: CP

PARACHUTE REFLEX:



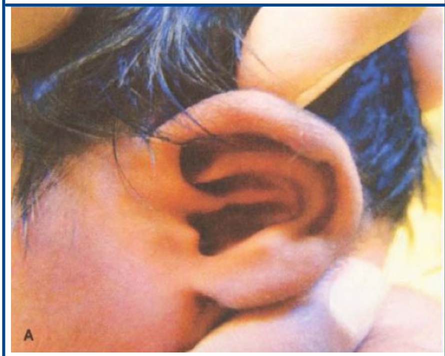
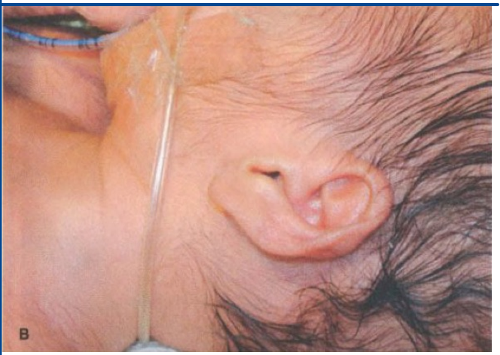
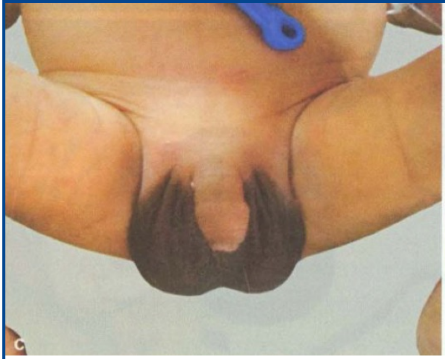
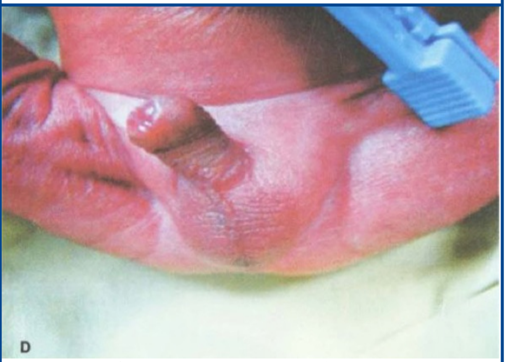
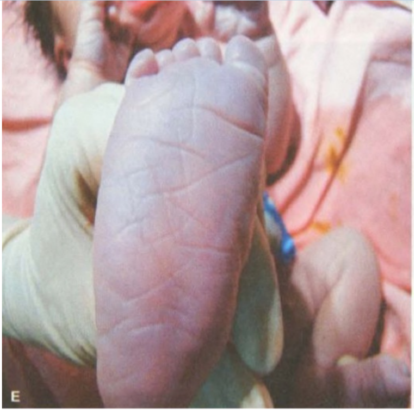
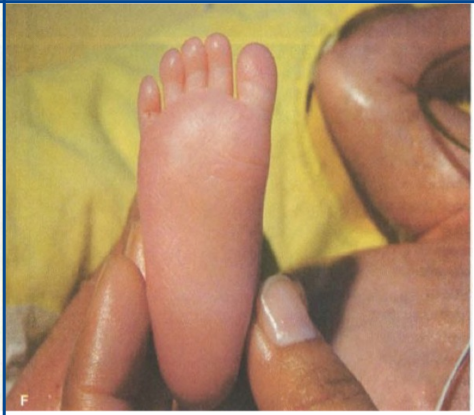
- Protective reflex
- Persistent throughout life
- Onset: 7-8 months after birth

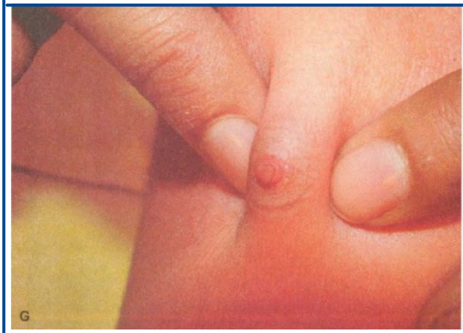
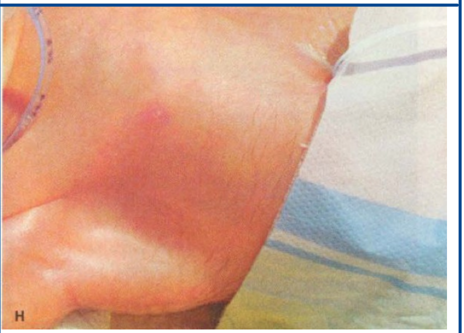
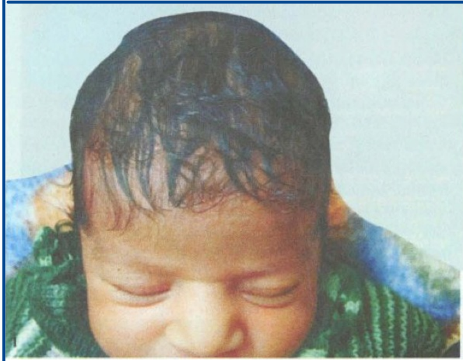
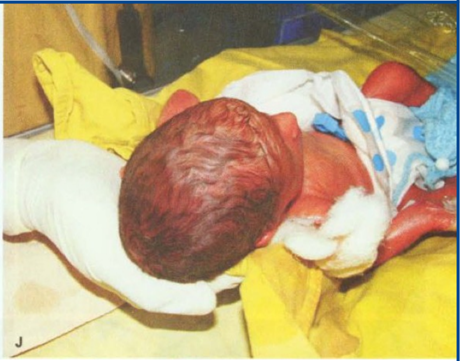
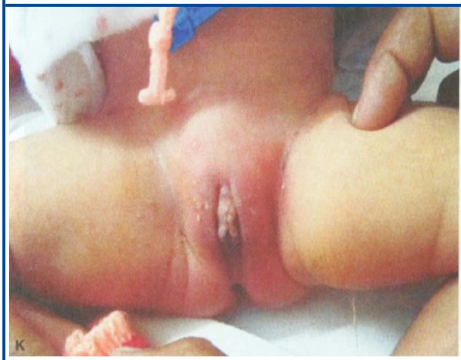
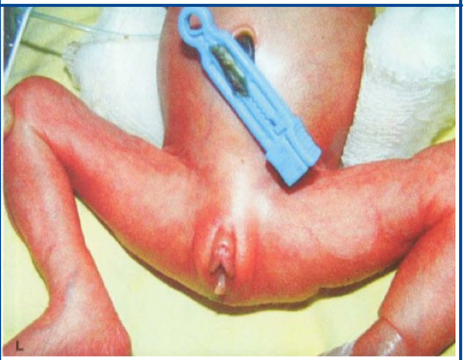
ROOTING REFLEX:

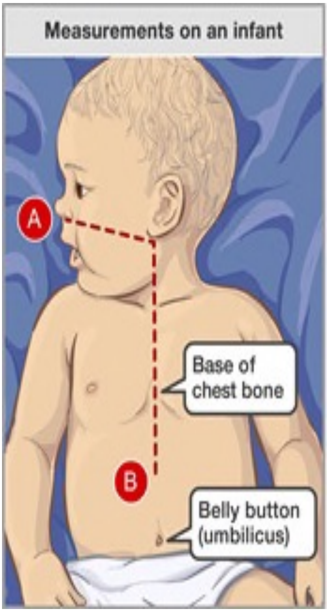
- Appears by 32 weeks of gestation
- Fully developed by 34 weeks
- Helps in breast feeding

NEONATAL REFLEX AFTER BIRTH:

- Symmetric tonic neck reflex
- Parachute reflex
- Landon reflex

TERM BABIES	PRETERM BABIES
 Well-curved pinna, cartilage reaching up to periphery	 Flat and soft pinna, cartilage not reaching up to periphery
 Well pigmented and pendulous scrotal sacs, with fully descended testes	 Light pigmentation and not yet descended testes
 Deep transverse creases on the soles	 Faint marks on the sole, no deep creases

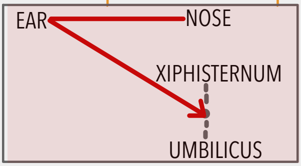
TERM BABIES	PRETERM BABIES
 Well formed breast bud (>5mm)	 Poorly developed breast bud
 Silky hair, where individual strands can be made out	 Fuzzy hair
 Labia majora covering clitoris and labia minora	 Prominent labia minora and clitoris



< 28 weeks: Give total parental nutrition (TPN)
Extreme premature
Low propulsive movement of GIT
Digestion impaired

TPN is also given if baby is hemodynamically unstable

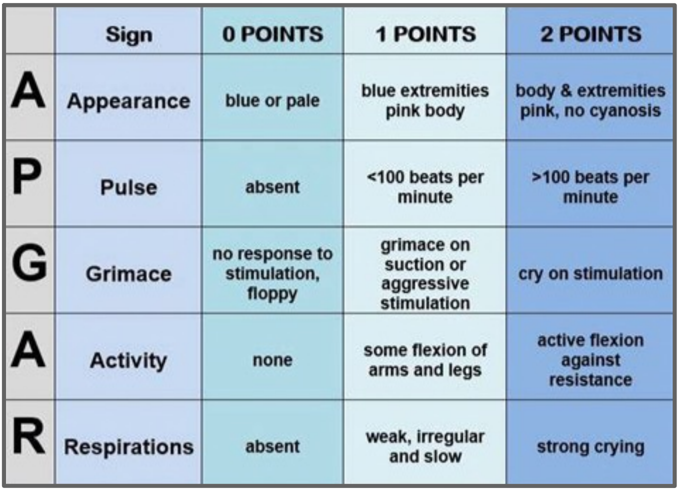
< 32 weeks: Expressed breast milk
(NG/ OG tube) - Tip of nose to tragus to midpoint between xiphisternum and umbilicus



32- 34 weeks: Expressed breast milk
- Palladian/ Katori spoon feeding

>34 weeks: Direct breast feeding

APGAR SCORE



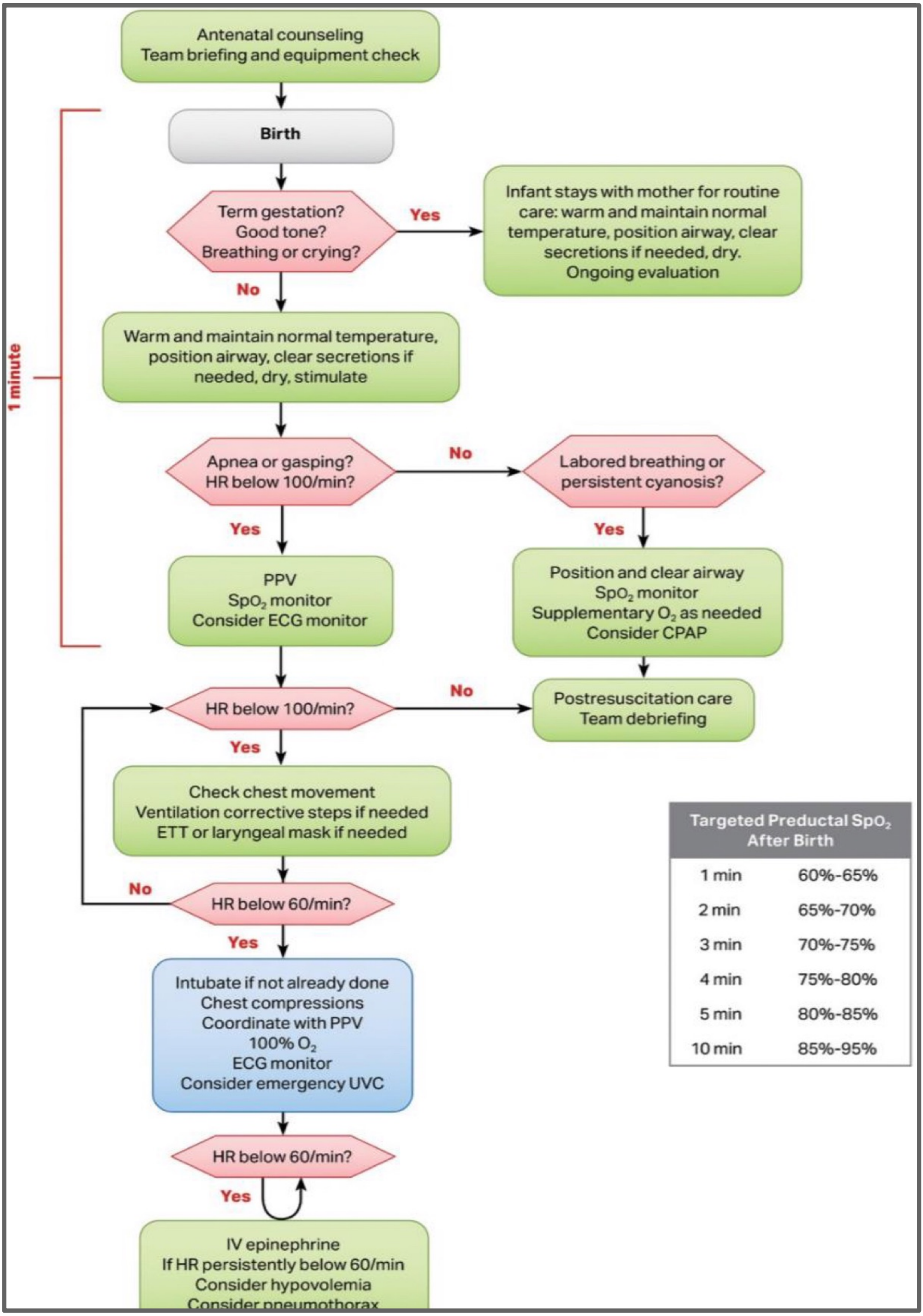
	Sign	0 POINTS	1 POINTS	2 POINTS
A	Appearance	blue or pale	blue extremities pink body	body & extremities pink, no cyanosis
P	Pulse	absent	<100 beats per minute	>100 beats per minute
G	Grimace	no response to stimulation, floppy	grimace on suction or aggressive stimulation	cry on stimulation
A	Activity	none	some flexion of arms and legs	active flexion against resistance
R	Respirations	absent	weak, irregular and slow	strong crying

Taken at 1min, 5min if required after 10mins

- Maximum score: 10
- Minimum score: 0
- Normal score: 8-10
- Low apgar score : <7 (Indicator of birth asphyxia)
- Severe asphyxia score: 0-3

[NEET 19]

APGAR score 3 at 1 minute indicates:


Antenatal counseling
Team briefing and equipment check
1 minute
Birth
Term gestation?
Good tone?
Breathing or crying?
Yes
Infant stays with mother for routine care: warm and maintain normal temperature, position airway, clear secretions if needed, dry.
Ongoing evaluation
No
Warm and maintain normal temperature, position airway, clear secretions if needed, dry, stimulate
Apnea or gasping?
HR below 100/min?
No
Labored breathing or persistent cyanosis?
Yes
PPV
SpO_2 monitor
Consider ECG monitor
Yes
Position and clear airway
SpO_2 monitor
Supplementary O_2 as needed
Consider CPAP
HR below 100/min?
No
Postresuscitation care
Team debriefing
Yes
Check chest movement
Ventilation corrective steps if needed
ETT or laryngeal mask if needed
No
HR below 60/min?
Yes
Intubate if not already done
Chest compressions
Coordinate with PPV
100% O_2
ECG monitor
Consider emergency UVC
HR below 60/min?
Yes
IV epinephrine
If HR persistently below 60/min
Consider hypovolemia
Consider pneumothorax

Targeted Preductal SpO_2 After Birth	
1 min	60%-65%
2 min	65%-70%
3 min	70%-75%
4 min	75%-80%
5 min	80%-85%
10 min	85%-95%

Main priority: Airway (respiration)
Most effective intervention in neonatal resuscitation : Bag and mask ventilation (PPV)
Positive pressure ventilation → ↑HR

Bag and mask ventilation : contraindicated in Congenital diaphragmatic hernia

MOST SENSIBLE INDICATOR OF HYPOXIA-> HR

RESPIRATORY DISTRESS

- Tachypnea (RR >60 per min) is accompanied by chest retractions and or grunt.

SCORING SYSTEM

SILVERMAN

- Preterm
- Maximum score: 10
- Minimum score : 0 (fully normal)
- > 6 – severe respiratory distress

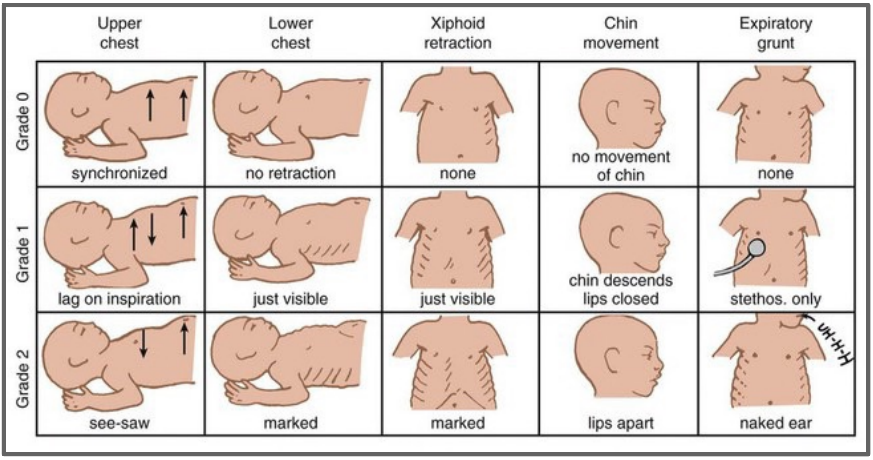
DOWNE

- All babies

DOWNE SCORING

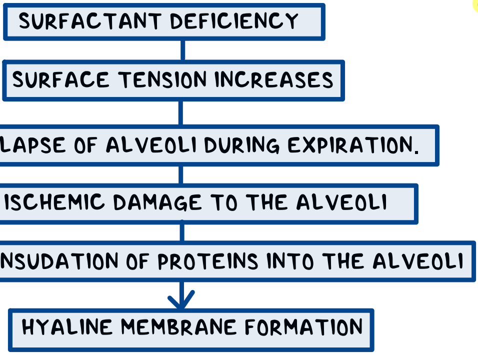
Score	Cyanosis	Respiratory rate	Air entry	Retractions	Grunting
0	Nil	<60/min	Normal	Nil	None
1	In room air	60 – 80/ min	Mild decrease	Mild	Audible with stethoscope
2	In 40% FiO2	>80/min	Marked decrease	Moderate - severe	Audible with naked ear

SILVERMAN SCORING


Upper chest
Lower chest
Xiphoid retraction
Chin movement
Expiratory grunt
Grade 0
synchronized
no retraction
none
no movement of chin
none
Grade 1
lag on inspiration
just visible
just visible
chin descends lips closed
stethos. only
Grade 2
see-saw
marked
marked
lips apart
H-H-Hn
naked ear

RESPIRATORY DISTRESS SYNDROME (HYALINE MEMBRANE DISEASE)

- Common in Preterm : < 34 weeks
- Respiratory distress begins within 6 hrs after birth



SURFACTANT DEFICIENCY

SURFACE TENSION INCREASES

COLLAPSE OF ALVEOLI DURING EXPIRATION.

ISCHEMIC DAMAGE TO THE ALVEOLI

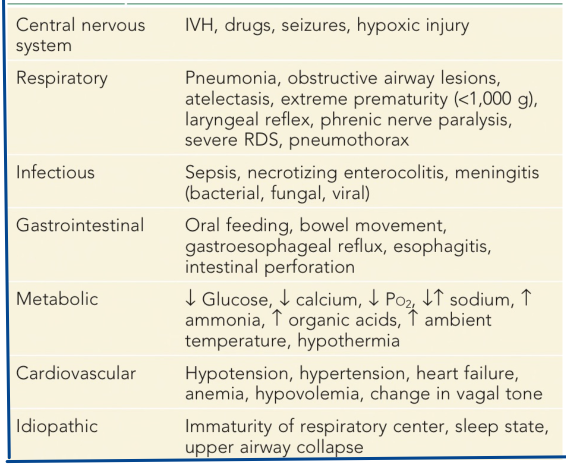
TRANSUDATION OF PROTEINS INTO THE ALVEOLI

HYALINE MEMBRANE FORMATION

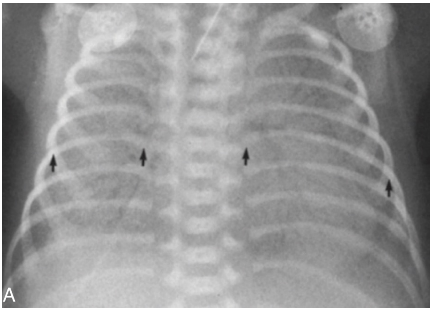
CLINICAL FEATURES

- Tachypnea,
- Retractions,
- Grunting,
- Cyanosis,
- Decreased air entry

POTENTIAL CAUSES OF NEONATAL APNEA



Central nervous system	IVH, drugs, seizures, hypoxic injury
Respiratory	Pneumonia, obstructive airway lesions, atelectasis, extreme prematurity (<1,000 g), laryngeal reflex, phrenic nerve paralysis, severe RDS, pneumothorax
Infectious	Sepsis, necrotizing enterocolitis, meningitis (bacterial, fungal, viral)
Gastrointestinal	Oral feeding, bowel movement, gastroesophageal reflux, esophagitis, intestinal perforation
Metabolic	↓ Glucose, ↓ calcium, ↓ PO_2, ↓↑ sodium, ↑ ammonia, ↑ organic acids, ↑ ambient temperature, hypothermia
Cardiovascular	Hypotension, hypertension, heart failure, anemia, hypovolemia, change in vagal tone
Idiopathic	Immaturity of respiratory center, sleep state, upper airway collapse



The infant is intubated, and the lungs show a dense reticulonodular pattern with air bronchograms.

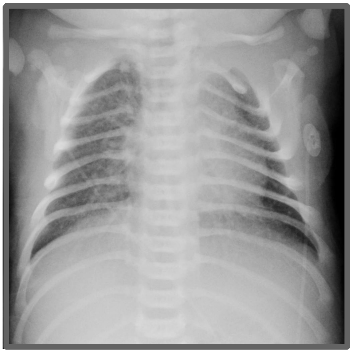
TRANSIENT TACHYPNEA OF NEWBORN

WET LUNG SYNDROME

On Chest X-ray

- Sun burst appearance
- Fluid in interlobar fissure

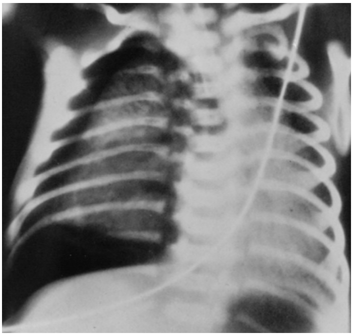
Improves within 72 hrs maximum



PNEUMOTHORAX

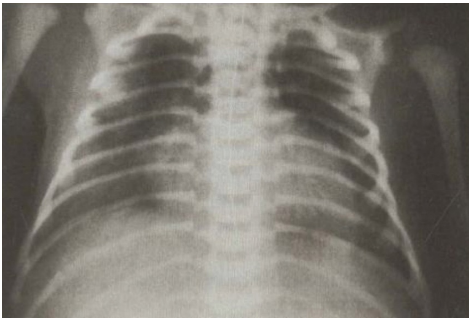
Alveolar rupture adjacent to the pleural space produces a pneumothorax .

If the gas is under tension, the pneumothorax shifts the mediastinum to the opposite side of the chest, producing hypotension, hypoxia, and hypercapnia.



MECONIUM ASPIRATION SYNDROME

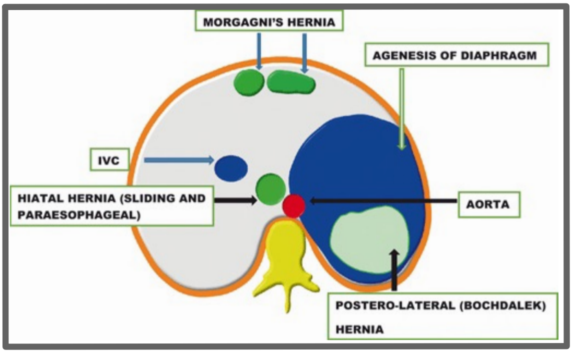
Aspirated meconium can block the large and small airway causing areas of atelectasis and emphysema which can progress to develop air leak syndromes like pneumothorax.

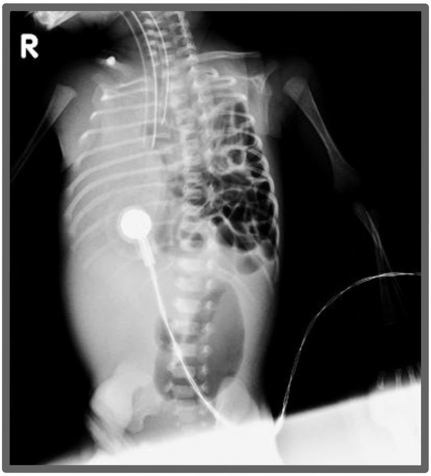


Hyperexpansion of lungs and heterogeneous opacities in right lung

CONGENITAL DIAPHRAGMATIC HERNIA

Failure of closure of the pleuroperitoneal membrane
↓
Intestinal loops ascend to the thorax
↓
Compress the developing lung
↓
PULMONARY HYPOPLASIA


MORGAGNI'S HERNIA
AGENESIS OF DIAPHRAGM
IVC
HIATAL HERNIA (SLIDING AND PARAESOPHAGEAL)
AORTA
POSTERO-LATERAL (BOCHDALEK) HERNIA



- Respiratory distress, scaphoid abdomen,
- Absent breath sound on left side
- Heart sound shifted to right,
- Bowel sounds on left side

Most common : Bochdalek hernia
(Posterior- lateral defect ; left side)

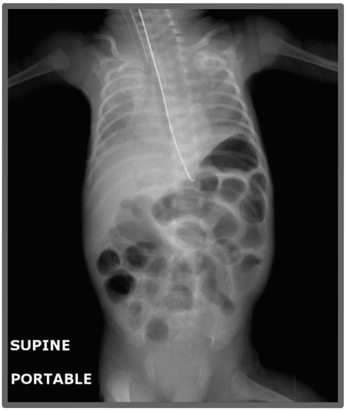
BAG AND MASK VENTILATION IS CONTRAINDICATED

NECROTISING ENTEROCOLITIS (NEC)

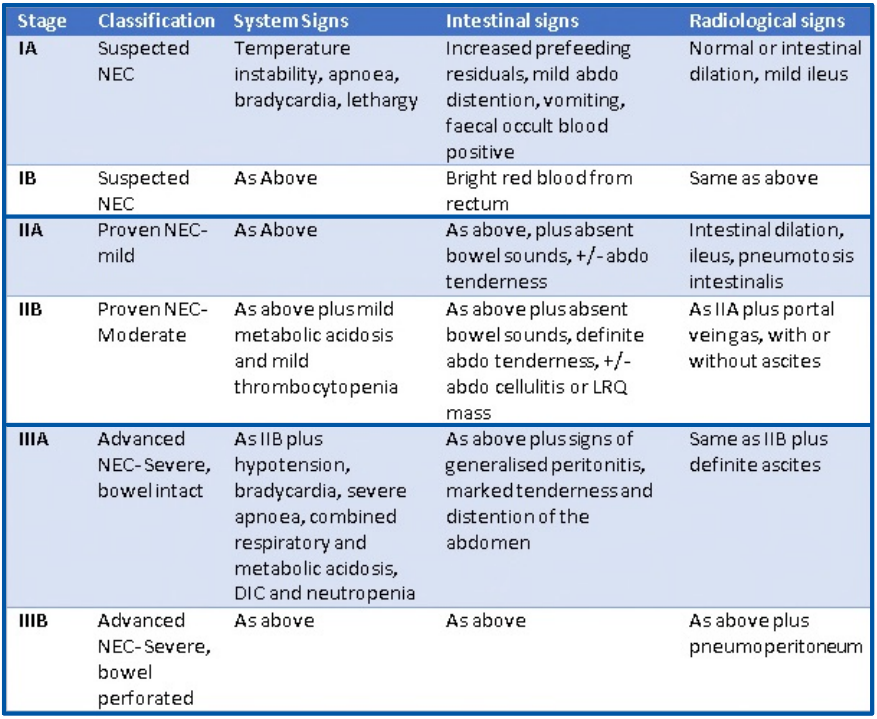
- Necrosis along with inflammation
- Result from interaction of several factors such as gut immaturity, mucosal injury due to hypoxia-ischemia, milk feeding and infection.

X-RAY FINDINGS

- PNEUMATOSIS (GAS CYSTS IN THE BOWEL WALL)
- DILATED LOOPS OF BOWEL
- PORTAL AIR
- FREE AIR (IF PERFORATED)


SUPINE
PORTABLE

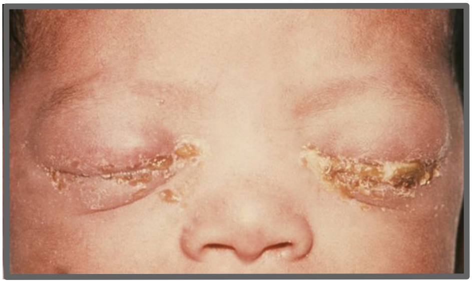
PNEUMATOSIS INTESTINALIS
- Air in intestine



Stage	Classification	System Signs	Intestinal signs	Radiological signs
IA	Suspected NEC	Temperature instability, apnoea, bradycardia, lethargy	Increased prefeeding residuals, mild abdo distention, vomiting, faecal occult blood positive	Normal or intestinal dilation, mild ileus
IB	Suspected NEC	As Above	Bright red blood from rectum	Same as above
IIA	Proven NEC- mild	As Above	As above, plus absent bowel sounds, +/- abdo tenderness	Intestinal dilation, ileus, pneumotosis intestinalis
IIB	Proven NEC- Moderate	As above plus mild metabolic acidosis and mild thrombocytopenia	As above plus absent bowel sounds, definite abdo tenderness, +/- abdo cellulitis or LRQ mass	As IIA plus portal vein gas, with or without ascites
IIIA	Advanced NEC- Severe, bowel intact	As IIB plus hypotension, bradycardia, severe apnoea, combined respiratory and metabolic acidosis, DIC and neutropenia	As above plus signs of generalised peritonitis, marked tenderness and distention of the abdomen	Same as IIB plus definite ascites
IIIB	Advanced NEC- Severe, bowel perforated	As above	As above	As above plus pneumoperitoneum

OPHTHALMIA NEONATORUM

Conjunctivitis occurring within the first month after birth



Gonococcal conjunctivitis
Most serious type of conjunctivitis in a newborn associated with vision impairment
Present on Day 2-5 (1st week)

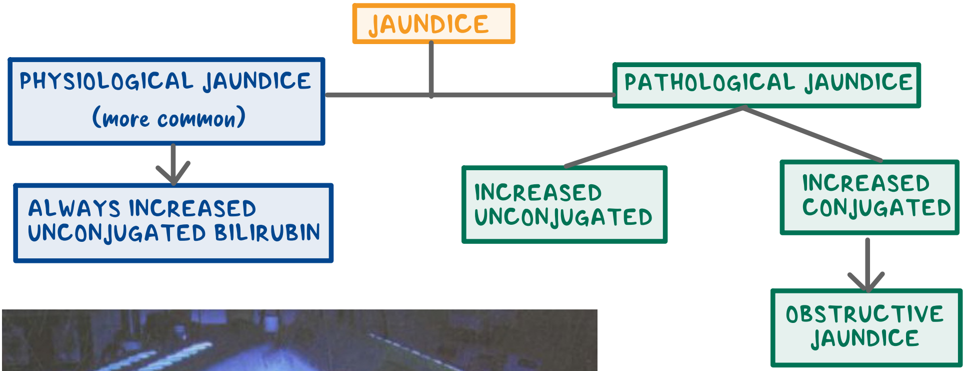
CLINICAL FEATURES

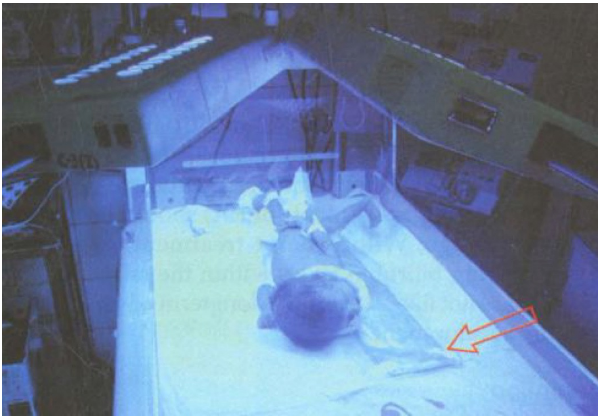
- PURULENT BILATERAL CONJUNTIVAL DISCHARGE
- HYPER ACUTE BLENORRHOEA
- SWELLING OF LIDS
- MUCOPURULENT DISCHARGE

JAUNDICE IN NEWBORN

VISIBLE JAUNDICE

Elevated bilirubin: > 5mg/dl


JAUNDICE
PHYSIOLOGICAL JAUNDICE (more common)
ALWAYS INCREASED UNCONJUGATED BILIRUBIN
PATHOLOGICAL JAUNDICE
INCREASED UNCONJUGATED
INCREASED CONJUGATED
OBSTRUCTIVE JAUNDICE

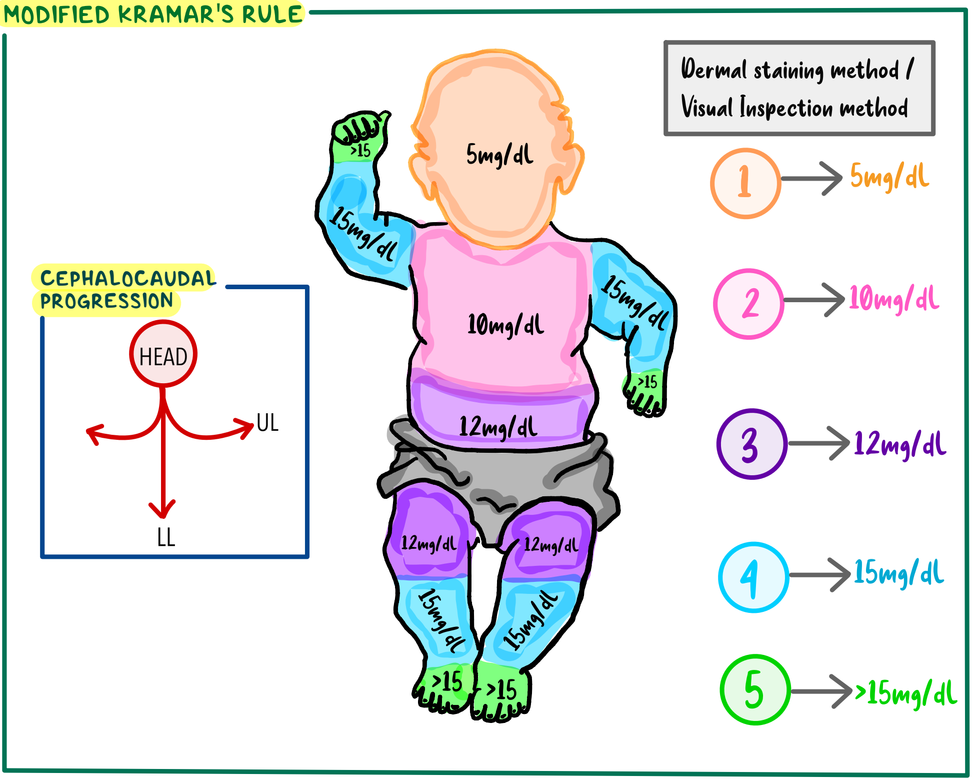


A jaundiced baby receiving phototherapy with two overhead units and biliblanket pad

PHYSIOLOGICAL JAUNDICE

- Always increased unconjugated bilirubin
- Appearance after 24hrs
- Duration
  - 2 weeks in preterm
  - 1 week in term
- KRAMMER'S rule:
  - Cephalocaudal progression
  - Does not involve palms and soles
- Rate of rise of bilirubin < 5mg/dl per 24 hrs

MODIFIED KRAMAR'S RULE


Dermal staining method / Visual Inspection method
CEPHALOCAUDAL PROGRESSION
HEAD
UL
LL
5mg/dl
10mg/dl
12mg/dl
15mg/dl
>15
12mg/dl
12mg/dl
15mg/dl
15mg/dl
>15
>15
>15
15mg/dl
1 → 5mg/dl
2 → 10mg/dl
3 → 12mg/dl
4 → 15mg/dl
5 → >15mg/dl

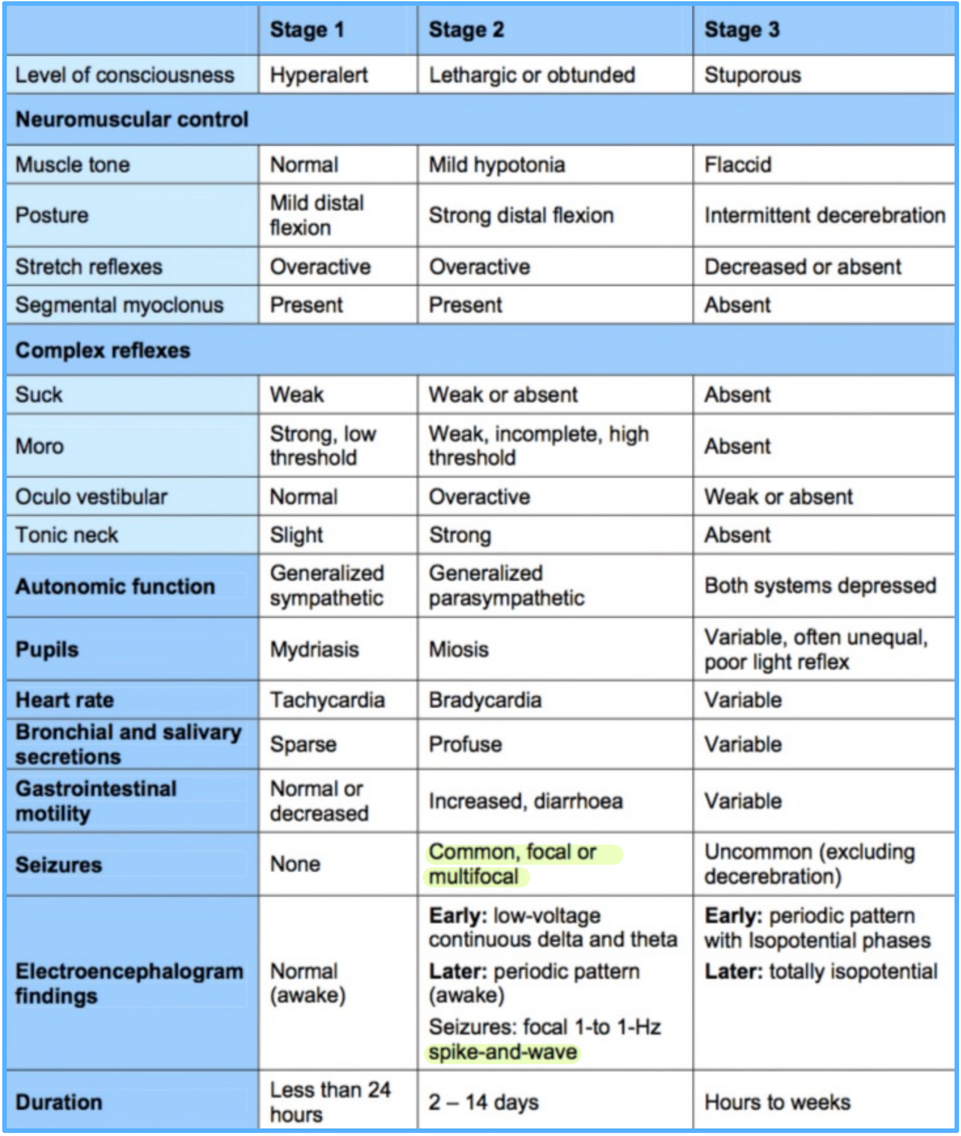
ZONE	APPROXIMATE TOTAL SERUM BILIRUBIN	
	MILD JAUNDICE (Lemon yellow skin)	DEEP JAUNDICE (Orange yellow skin)
①	5-7mg/dl	7-9mg/dl
②	7-9 mg/dl	9-11mg/dl
③	9-11mg/dl	11-13mg/dl
④	11-13mg/dl	13-16mg/dl
⑤	13-15mg/dl	17mg/dl

HYPOXIC ISCHEMIC ENCEPHALOPATHY

- Hypoxia affecting the brain
- Most common organ affected by birth asphyxia - brain

SARNAT AND SARNAT STAGING FOR HIE

Mild Moderate Severe



	Stage 1	Stage 2	Stage 3
Level of consciousness	Hyperalert	Lethargic or obtunded	Stuporous
Neuromuscular control			
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive	Decreased or absent
Segmental myoclonus	Present	Present	Absent
Complex reflexes			
Suck	Weak	Weak or absent	Absent
Moro	Strong, low threshold	Weak, incomplete, high threshold	Absent
Oculo vestibular	Normal	Overactive	Weak or absent
Tonic neck	Slight	Strong	Absent
Autonomic function	Generalized sympathetic	Generalized parasympathetic	Both systems depressed
Pupils	Mydriasis	Miosis	Variable, often unequal, poor light reflex
Heart rate	Tachycardia	Bradycardia	Variable
Bronchial and salivary secretions	Sparse	Profuse	Variable
Gastrointestinal motility	Normal or decreased	Increased, diarrhoea	Variable
Seizures	None	Common, focal or multifocal	Uncommon (excluding decerebration)
Electroencephalogram findings	Normal (awake)	**Early:** low-voltage continuous delta and theta **Later:** periodic pattern (awake) Seizures: focal 1-to 1-Hz spike-and-wave	**Early:** periodic pattern with Isopotential phases **Later:** totally isopotential
Duration	Less than 24 hours	2 – 14 days	Hours to weeks

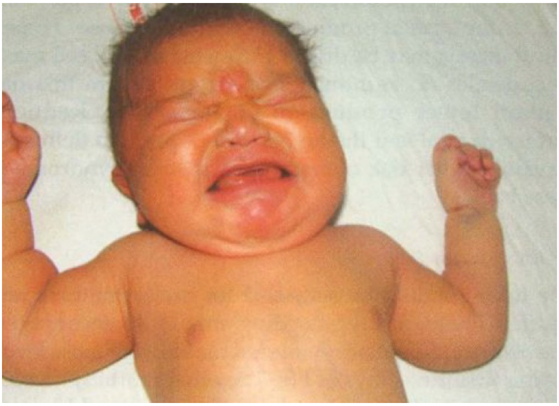
SEIZURE - STAGE II OF HIE

INFANT OF DIABETIC MOTHER

↓

FETAL HYPERINSULINEMIA

1stTrimester	↑ Risk of congenital Heart Disease and other organ systems
2nd Trimester	↑Risk of Abortion , still Birth , Preterm Labour
3rd Trimester	↓Surfactant production → HMD ↑RBC mass → Polycythemia ↑Bilirubin → NNJ ↑Weight gain → Macrosomia



Large size of the baby with broad shoulders and torso and a relatively smaller head

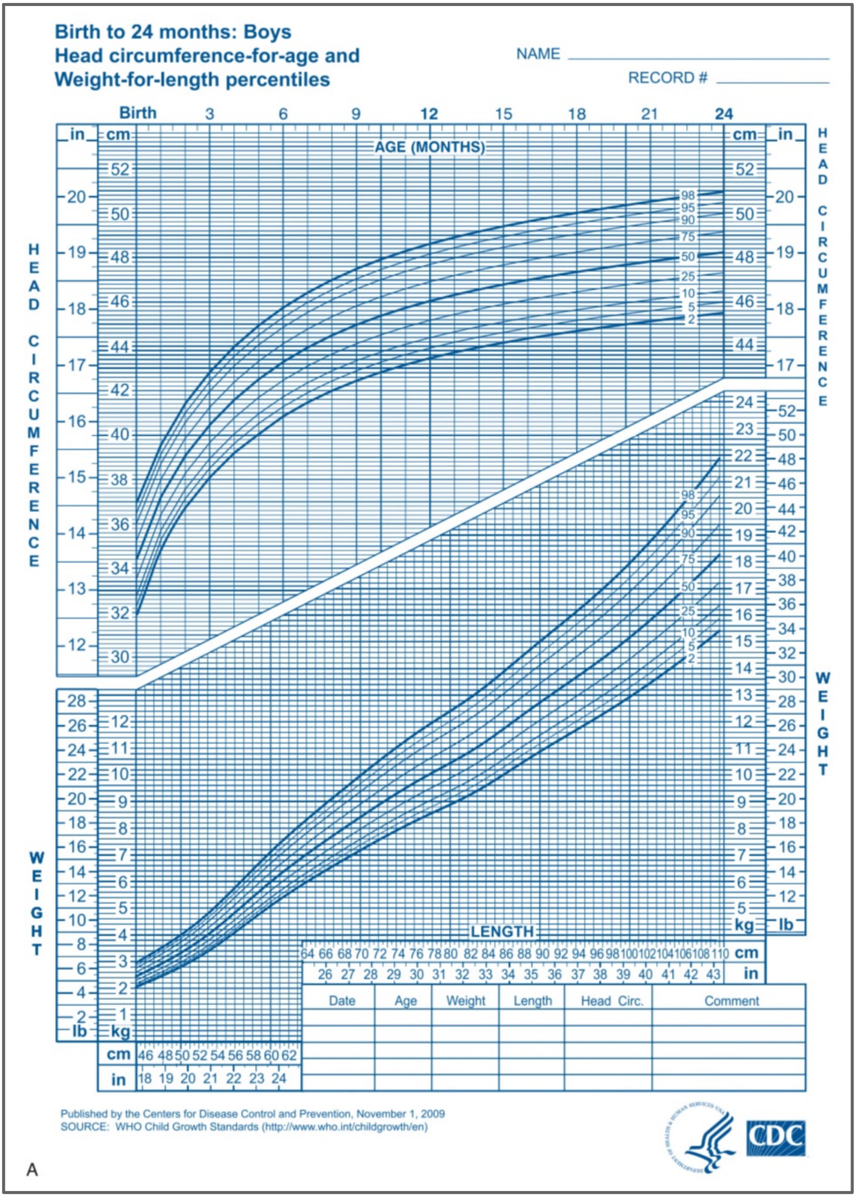
Most common NTD in IDM → Anencephaly

Most specific malformation in IDM → Caudal Regression syndrome

Most specific CVS malformation in IDM → TGA

Most common GIT abnormality in IDM → Duodenal Atresia > Hirschsprun'sg Disease .

WORLD HEALTH ORGANIZATION GROWTH CHARTS



Birth to 24 months: Boys
Head circumference-for-age and
Weight-for-length percentiles

NAME ______________________

RECORD # __________________

Date	Age	Weight	Length	Head Circ.	Comment

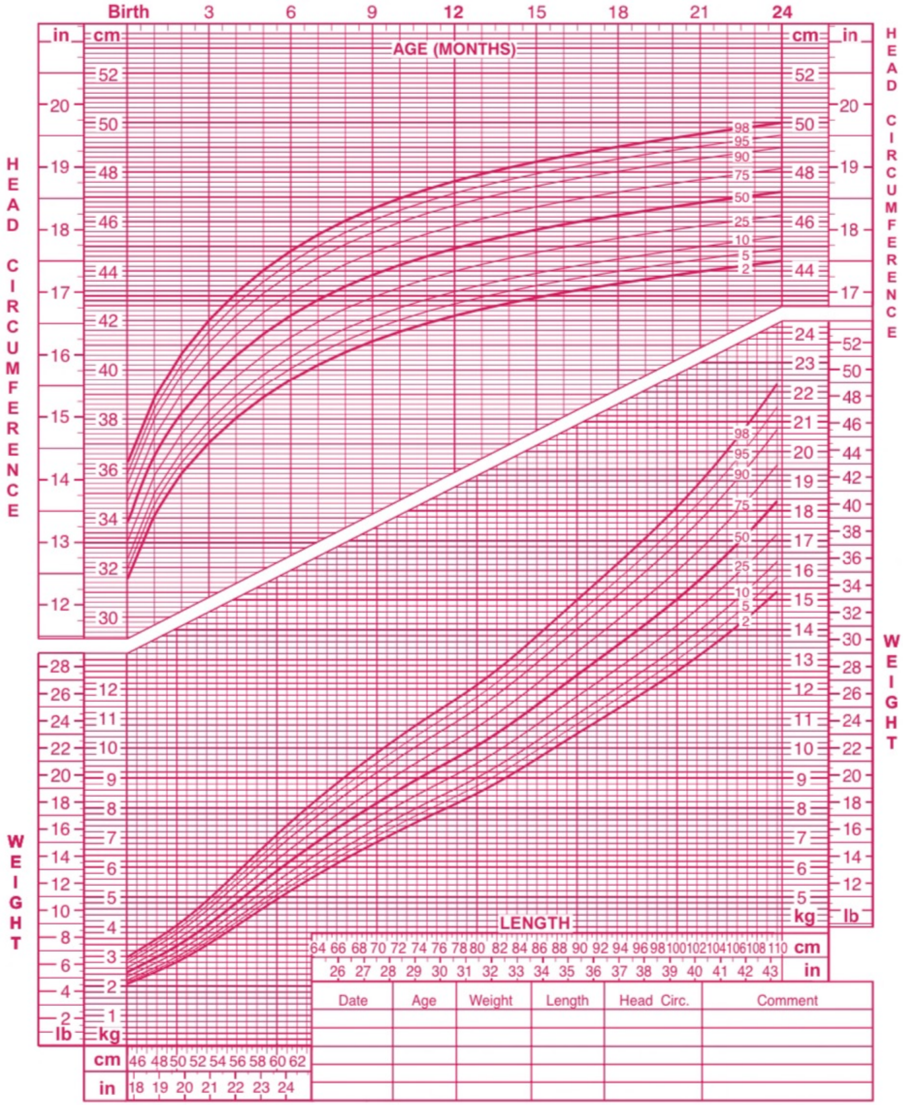
Published by the Centers for Disease Control and Prevention, November 1, 2009
SOURCE: WHO Child Growth Standards (http://www.who.int/childgrowth/en)

Reference: Essential Pediatrics Ghai (Pg 7,42)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 11)

Birth to 24 months: Girls
Head circumference-for-age and Weight-for-length percentiles

NAME ____________________

RECORD # __________



Date	Age	Weight	Length	Head Circ.	Comment

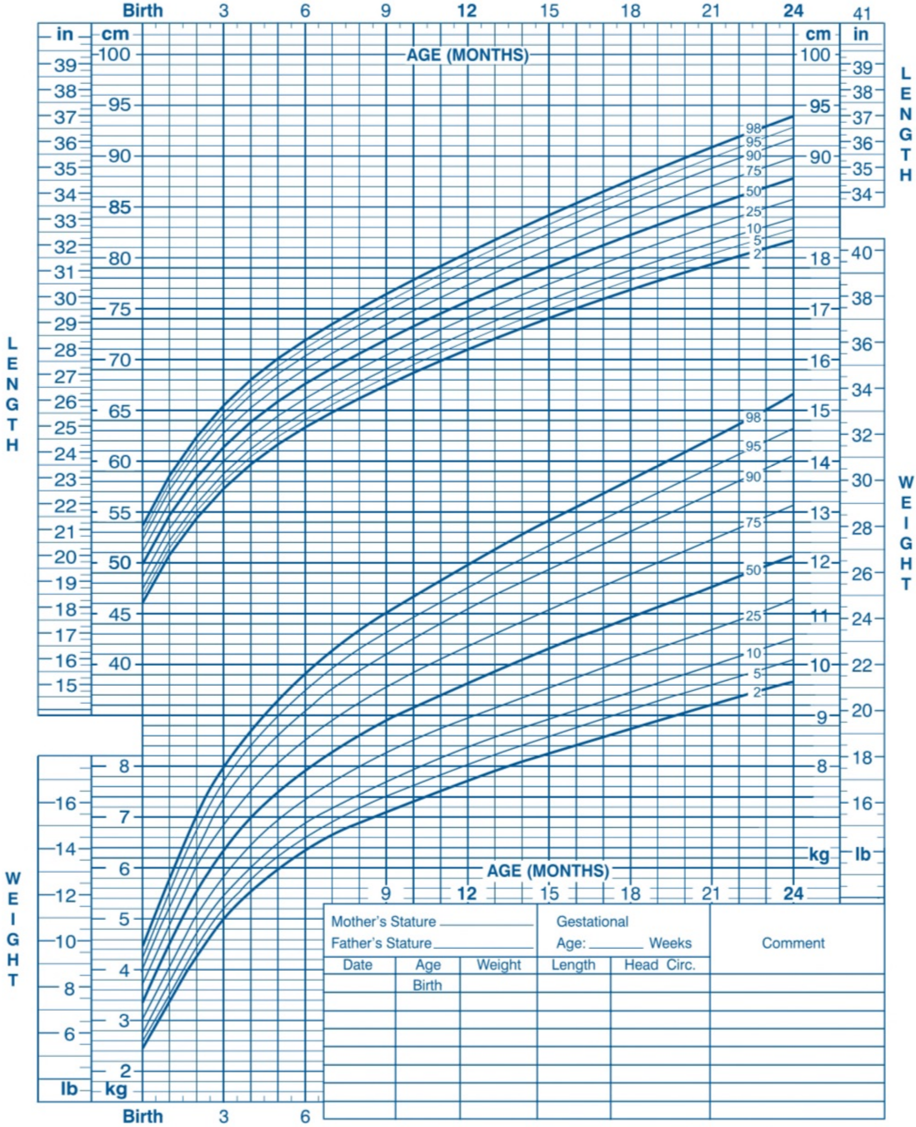
Published by the Centers for Disease Control and Prevention, November 1, 2009
SOURCE: WHO Child Growth Standards (http://www.who.int/childgrowth/en)

B

Birth to 24 months: Boys
Length-for-age and Weight-for-age percentiles

NAME ______________________

RECORD # ______________



Mother's Stature ______ Father's Stature ______			Gestational Age: ______ Weeks		Comment
Date	Age	Weight	Length	Head Circ.	
	Birth				

Published by the Centers for Disease Control and Prevention, November 1, 2009
SOURCE: WHO Child Growth Standards (http://www.who.int/childgrowth/en)

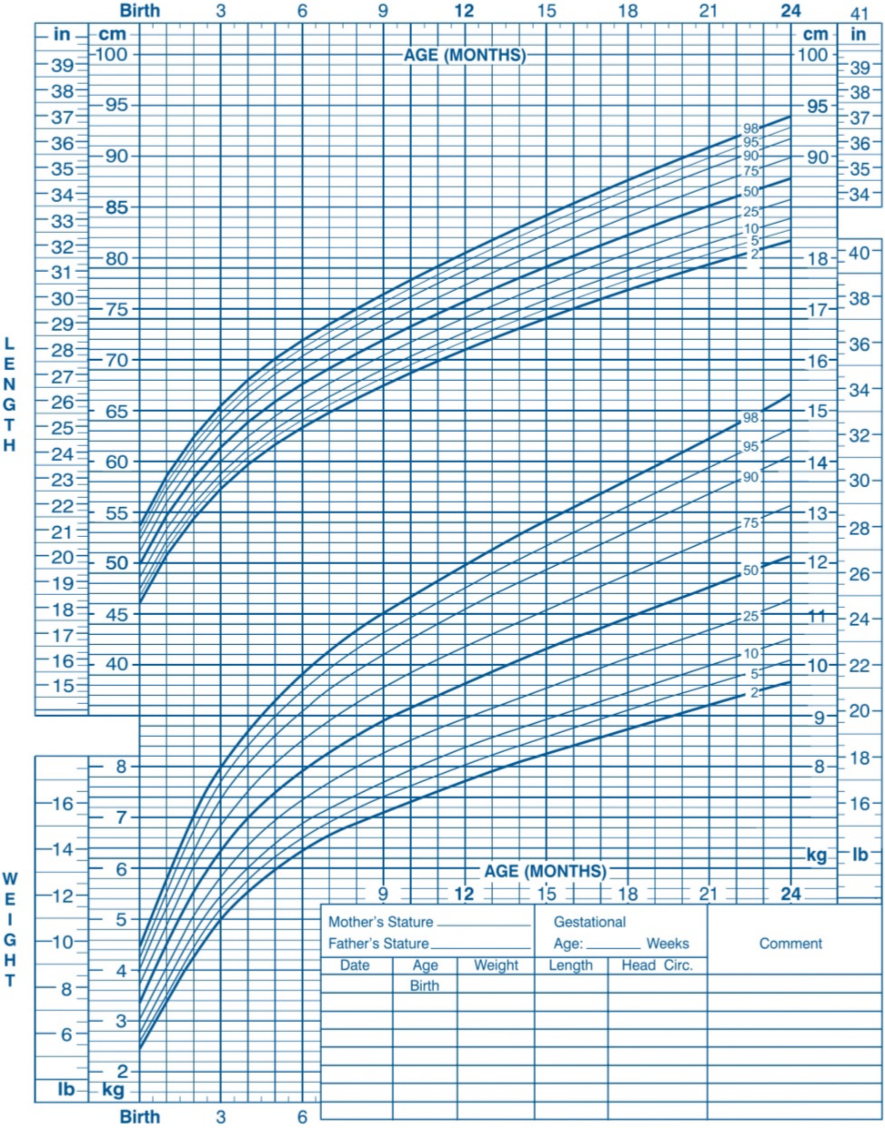
SAFER·HEALTHIER·PEOPLE™

A

Birth to 24 months: Boys
Length-for-age and Weight-for-age percentiles

NAME ____________________

RECORD # ____________



Mother's Stature ____________

Father's Stature ____________

Gestational Age: ________ Weeks

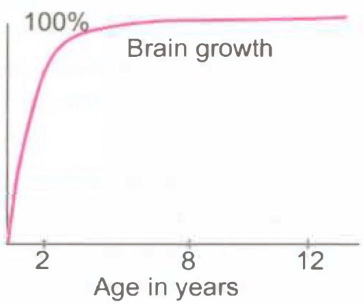
Date	Age	Weight	Length	Head Circ.	Comment
	Birth				

Published by the Centers for Disease Control and Prevention, November 1, 2009
SOURCE: WHO Child Growth Standards (http://www.who.int/childgrowth/en)

SAFER·HEALTHIER·PEOPLE™

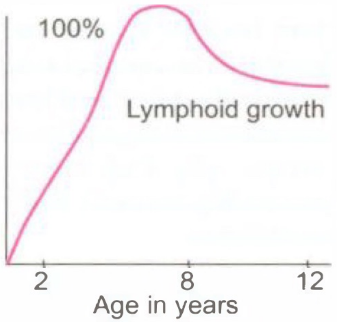
A

RATES OF GROWTH OF DIFFERENT TISSUES AND ORGANS



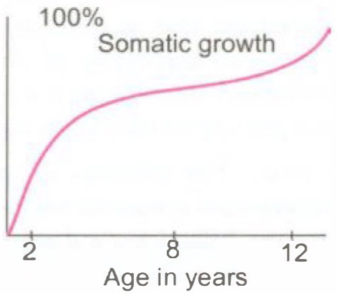
BRAIN GROWTH

- Brain enlarges rapidly during the latter months of fetal life and early months of postnatal life.
- At birth, the head size is about 65-70% of the expected head size in adults.
- It reaches 90% of the adult head size by the age of 2 years.



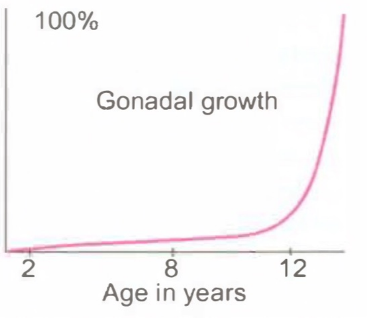
LYMPHOID GROWTH

- The growth of lymphoid tissue is most notable during mid-childhood.
- During this period, the lymphoid tissue is overgrown and its mass may appear to be larger than that of the fully mature adult.
- A sign of accelerated lymphoid growth is the frequent finding of large tonsils and palpable lymph nodes in normal children between 4 and 8 years.



SOMATIC GROWTH

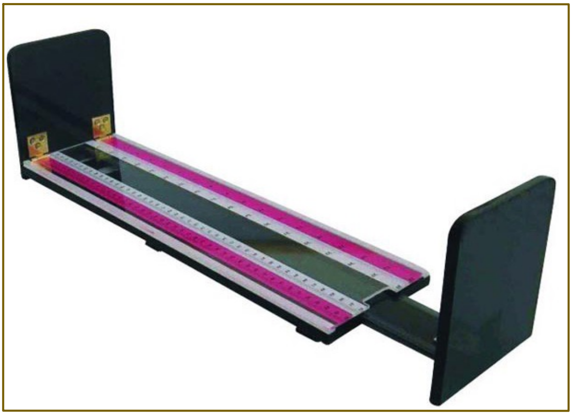
- Skeletal growth
- The skeleton is mature once the epiphysis or growth plates at the end of long bones fuse to the shaft or diaphysis.
- This occurs by about 18 years in girls and 20-22 years in boys.
- The degree of skeletal maturation closely correlates with the degree of sexual maturation.



GROWTH OF GONADS

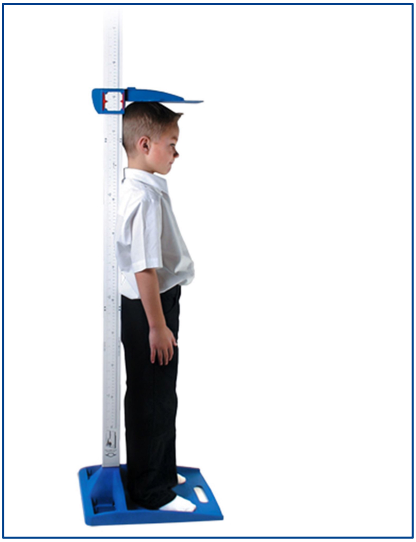
- Gonadal growth is dormant during childhood and becomes conspicuous during pubescence

ASSESSMENT OF PHYSICAL GROWTH

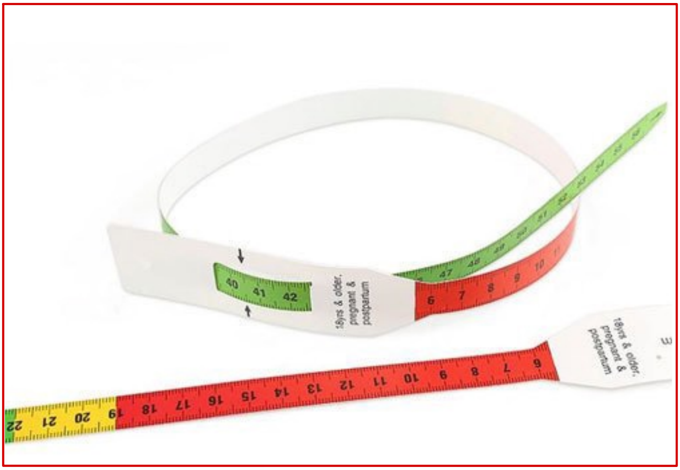


Infantometer

- Infantometer is a horizontal rod with a ruler, used to measure the length of an infant

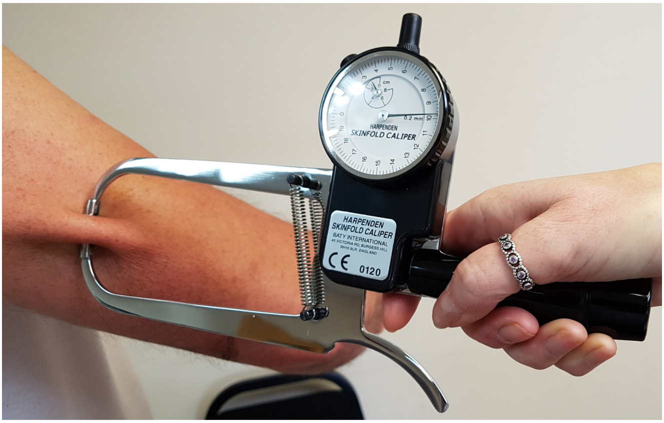


Stadiometer


18yrs & older, pregnant & postpartum

Sakir tape

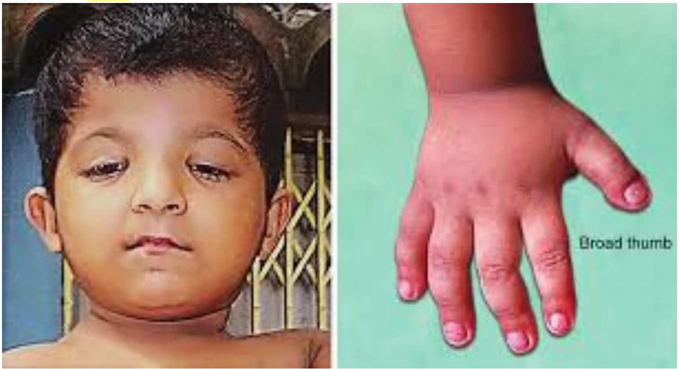
- A measuring tape with cm markings (6 to 26 cm) with 3 colored zones (red, yellow and green), used to measure the mid-arm circumference


HARPENDEN SKINFOLD CALIPER
BATY INTERNATIONAL
0120

Herpenden calliper

- Skin fold thickness is measured by Herpenden calliper

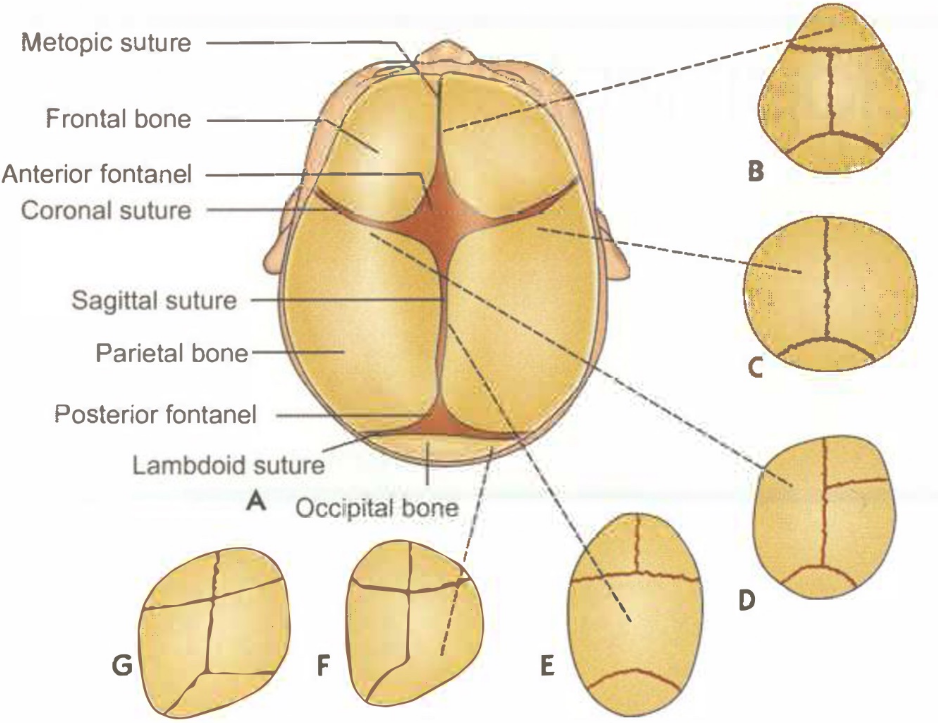
RUBINSTEIN-TAYBI SYNDROME


Broad thumb

i. Short stature
ii. Beaked nose
iii. Highly arched eyebrows
iv. Retrognathia
v. Broad thumbs
vi. Great toes which are deviated medially

Reference : https://www.ijopmr.com/doi/IJOPMR/pdf/10.5005/jp-journals-10066-0007

ABNORMALITIES OF HEAD


Metopic suture
Frontal bone
Anterior fontanel
Coronal suture
Sagittal suture
Parietal bone
Posterior fontanel
Lambdoid suture
A
Occipital bone
B
C
D
E
F
G

(A) Head of normal neonate showing fontanelles and sutures.

The common forms of craniosynostosis (secondary to premature fusion of sutures) include;

(B) trigonocephaly (metopic suture);
(C) brachycephaly (bilateral coronal sutures);
(D) left anterior plagiocephaly (left coronal suture);
(E) scaphocephaly (sagittal suture);
(F) right posterior plagiocephaly (right lambdoid suture) Children with deformation plagiocephaly;
(G) have positional skull flattening without sutural fusion

Ghai Essential Pediatrics 8th Edition, pg 37

SHORT STATURE

- < -2 SD height / age or < 3 rd percentile
- Normal variant > > pathological variant
- Most common cause of short stature or delayed puberty- Constitutional delay

NORMAL VARIANT

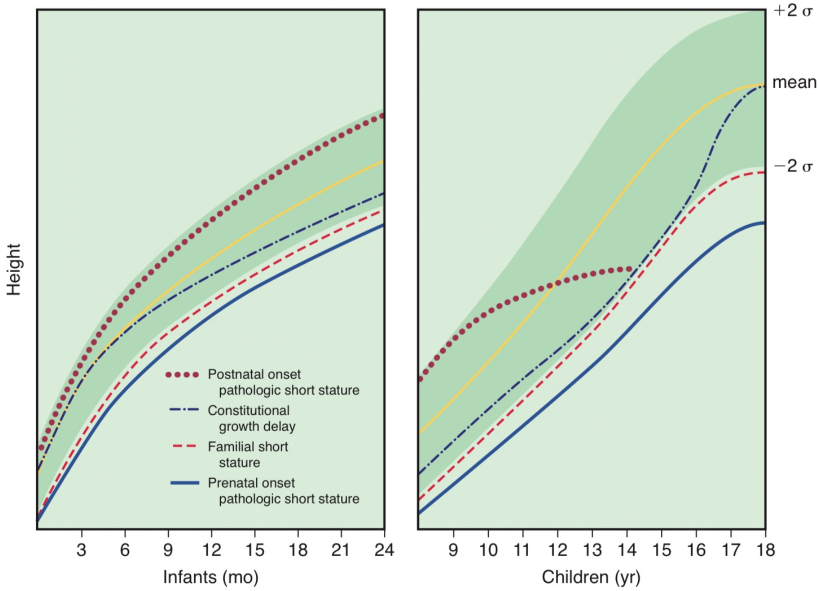
Growth velocity - rate of increase of height is normal

FAMILIAL/ GENETIC SHORT STATURE

- FINAL HEIGHT -SHORT
- PUBERTY TIME - NORMAL
- BONE AGE - MATCHES WITH ACTUAL AGE

CONSTITUTIONAL DELAY(Dip)

- FINAL HEIGHT - NORMAL
- PUBERTY TIME - DELAYED
- BONE AGE - LESS THAN ACTUAL AGE

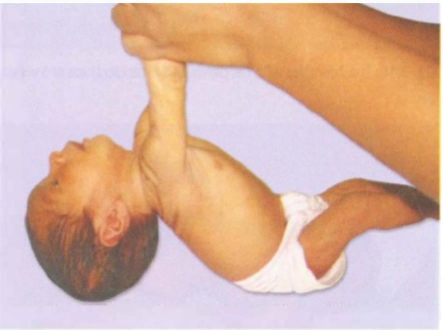


DEVELOPMENTAL MILESTONES

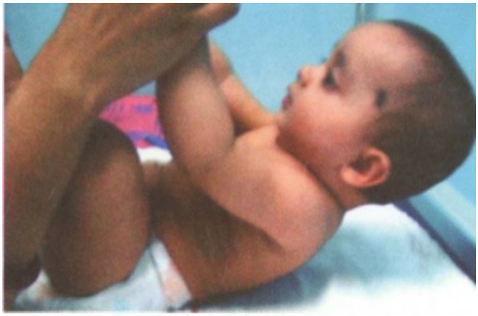
KEY GROSS MOTOR DEVELOPMENTAL MILESTONES

AGE	MILESTONE
3 Months	NECK HOLDING
5 Months	ROLLS OVER
6 Months	SITS IN TRIPOD FASHION (SITTING WITH OWN SUPPORT)
8 Months	SITTING WITHOUT SUPPORT
9 Months	STANDS HOLDING ON (WITH SUPPORT)
12 Months	CREEPS WELL; WALKS BUT FALLS; STANDS WITHOUT SUPPORT
15 Months	WALKS ALONE; CREEPS UPSTAIRS
18 Months	RUNS; EXPLORES DRAWERS
2 years	WALKS UP AND DOWNSTAIRS (2 FEET/STEP); JUMPS
3 years	RIDES TRICYCLE; ALTERNATE FEET GOING UPSTAIRS
4 years	HOPS ON FOOT; ALTERNATE FEET GOING DOWNSTAIRS

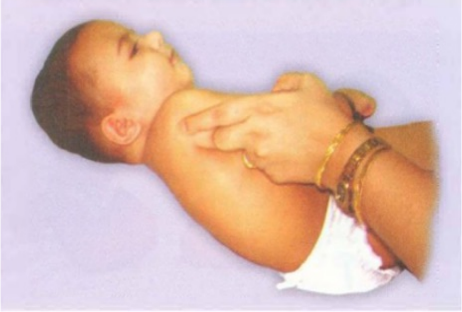
GROSS MOTOR DEVELOPMENT



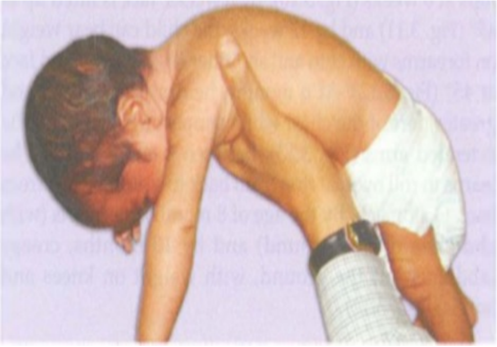
Pull to sit; complete head lag in a newborn



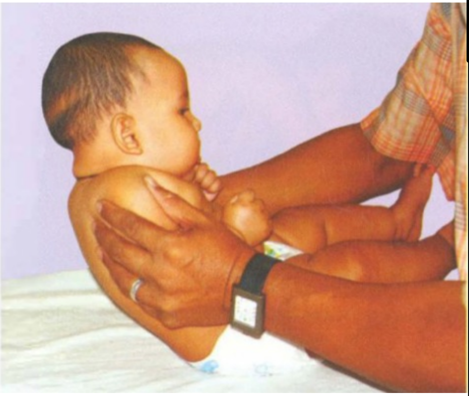
Infant lifts head from the supine position when about to be pulled at 5 months



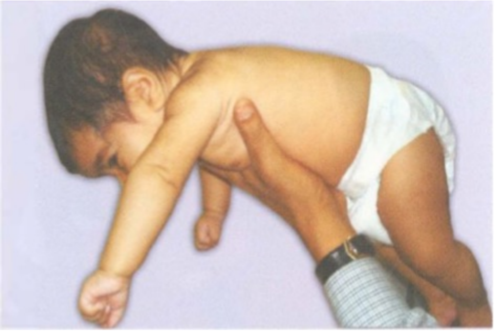
Pull to sit; no head lag at 4 months



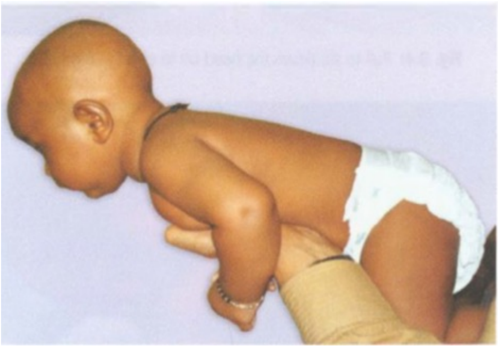
Ventral suspension; unable to hold neck in the line with trunk at 4 weeks



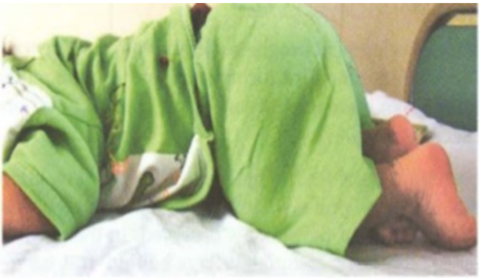
Pull to sit; flexes the head on to chest at 5 months



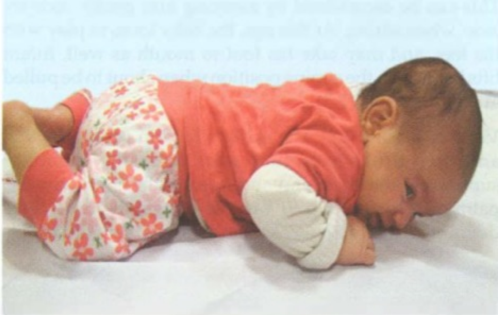
Ventral suspension; head in line with the trunk at 8 - 10 weeks



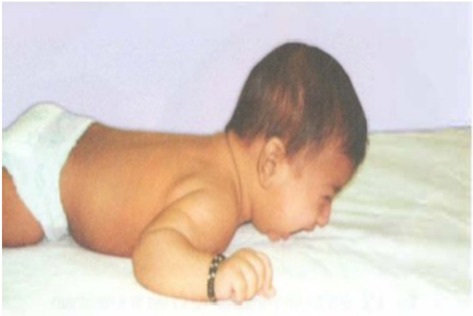
Ventral suspension; head in line with the trunk at 12 weeks



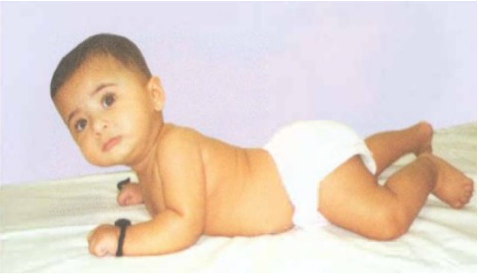
The infant lies on the bed with high pelvis and knees drawn up at 2 weeks



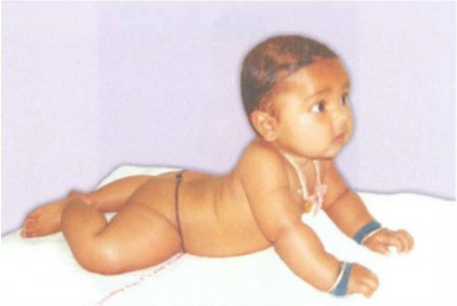
The infant lies with flat pelvis and extended hips at 6 weeks



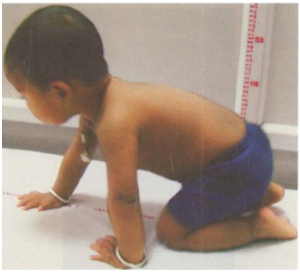
In prone: face lifted to about 45° at 8 weeks



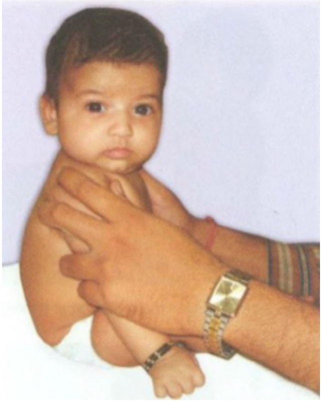
In prone: face, head and chest off the couch at 3 months



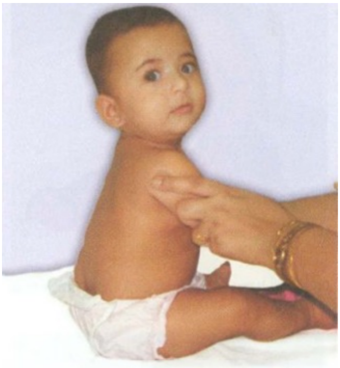
In prone: weight on hands with extended arms at 6 months



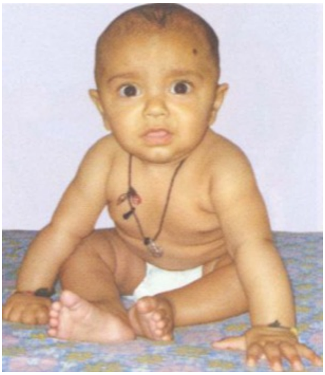
Creep position at 10 months of age (abdomen off ground and weight on hands and knees



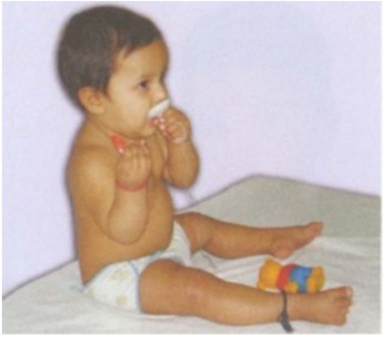
Sitting; back rounded but able to hold head at 8 weeks



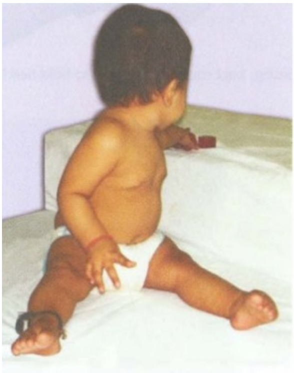
Sitting; back much straighter at 4 months



Sitting with support of hands at 6 months



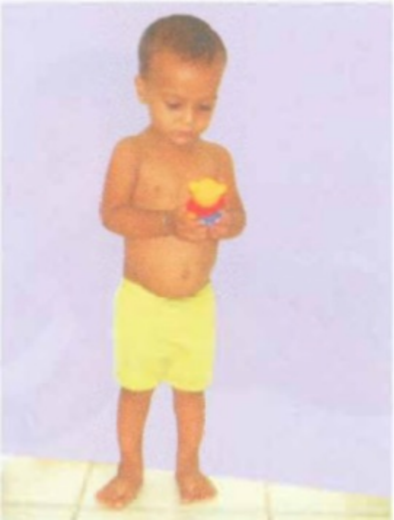
Sitting without support at 8 months



Pivoting; turns around to pick up an object at 11 months

Bears almost entire weight at 6 months

Child walking with one hand-held at 12-13 months



Stands well at 12 months

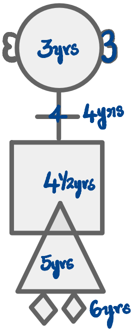
The child is able to walk upstairs and downstairs one foot per step at 4 yr

KEY FINE MOTOR DEVELOPMENTAL MILESTONES

AGE	MILESTONE
4 Months	BIDEXTROUS REACH (REACHING OUT FOR OBJECTS WITH BOTH HANDS)
6 Months	UNIDEXTROUS REACH (REACHING OUT FOR OBJECTS WITH ONE HAND); TRANSFER OBJECTS
9 Months	IMMATURE PINCER GRASP; PROBES WITH FOREFINGER
12 Months	PINCER GRASP MATURE
15 Months	IMITATES SCRIBBLING; TOWER OF 2 BLOCKS
18 Months	SCRIBBLES; TOWER OF 3 BLOCKS
2 years	TOWER OF 6 BLOCKS; VERTICAL AND CIRCULAR STROKE
3 years	TOWER OF 9 BLOCKS; COPIES CIRCLE
4 years	COPIES CROSS; BRIDGE WITH BLOCKS
5 years	COPIES TRIANGLE; GATE WITH BLOCKS

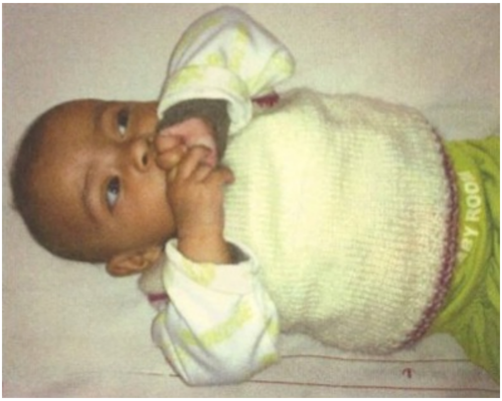
TOWER OF CUBES

3 yrs = (3x 3) = 9 cubes
2 yrs = (2x 3) = 6 cubes
8 months = 3 cubes
15 months= 2 cubes

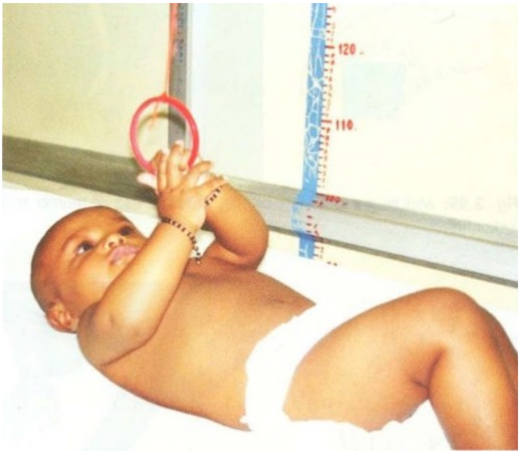


3 yrs- Circle
4 yrs - X/+
4 1/2 yrs- Square
5 yrs- Triangle
6 yrs - Diamond

Hand regard (between 12 and 20 weeks)



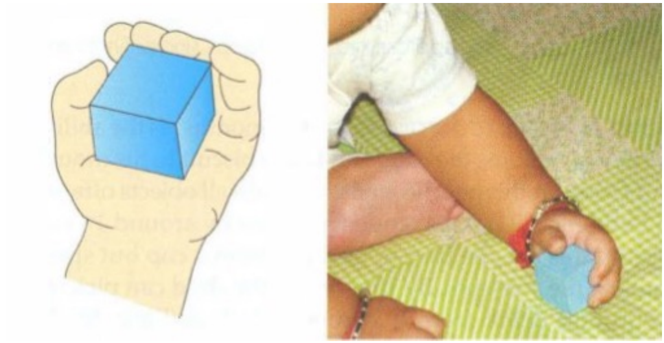
The child brings hands in midline as he plays at 3 to 4 months of age



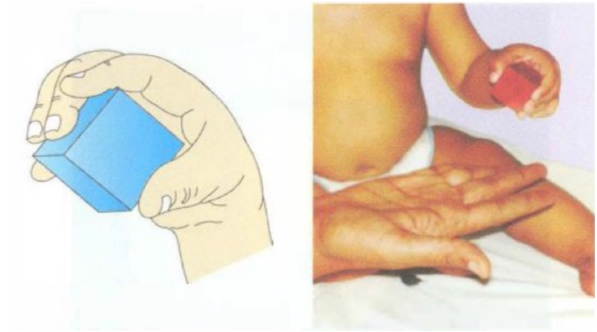
Bidextrous grasp approach to a dangling ring at 4 months

[NEET'19]

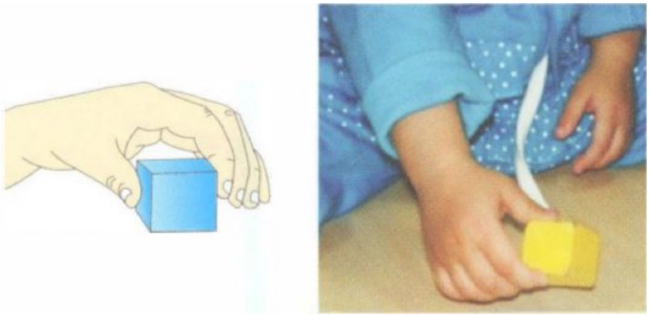
Bidextrous grip is seen at what age?



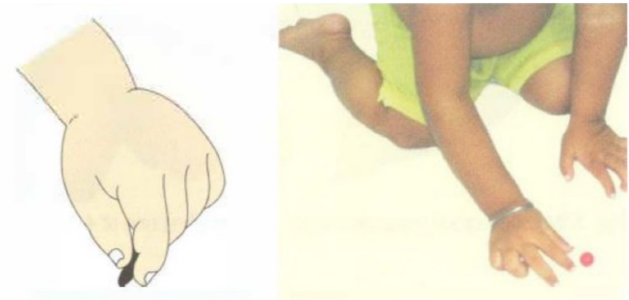
Immature grasp at 6 months (palmar grasp)



Intermediate grasp at 8 months, beginning to use radial aspect of the hand



Mature grasp at 1 yr of age, note the use of thumb and index finger



Pincer grasp approach to small objects (index finger and thumb)

A child mouthing an object at 6 months of age

Scribbles spontaneously at 15 months

A child makes tower of 5-6 cubes at 2 yr of age

KEY SOCIAL AND ADAPTIVE MILESTONES

AGE	MILESTONE
2 Months	SOCIAL SMILE (SMILE AFTER BEING TALKED TO)
3 Months	RECOGNIZES MOTHER; ANTICIPATES FEEDS
6 Months	RECOGNIZES STRANGERS, STRANGER ANXIETY
9 Months	WAVES "BYE BYE"
12 Months	COMES WHEN CALLED; PLAYS SIMPLE BALL GAME
15 Months	JARGON
18 Months	COPIES PARENTS IN TASK (E.G. SWEEPING)
2 years	ASKS FOR FOOD, DRINK, TOILET, PULLS PEOPLE TO SHOW TOYS
3 years	SHARES TOYS; KNOWS FULL NAME AND GENDER
4 years	PLAYS COOPERATIVELY IN A GROUP; GOES TO TOILET ALONE
5 years	HELPS IN HOUSEHOLD TASKS. DRESSES AND UNDRESSES

At 1 month, the baby showing intent regard of his mother's face as she talks to him

Social smile

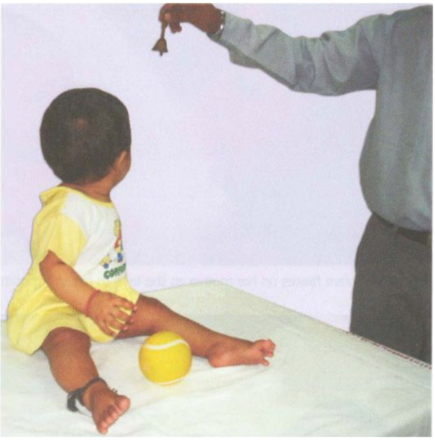
A child smiles at himself in the mirror at 6 months of age

KEY LANGUAGE MILESTONES

AGE	MILESTONES
1 Months	ALERTS TO SOUND
3 Months	COOS (MUSICAL VOWEL SOUND)
4 Months	LAUGH LOUD
6 Months	MONOSYLLABLES (BA, DA, PA). AH-GOO SOULD
9 Months	BISYLLABLES (MAMA, DADA, BABA)
12 Months	1-2 WORDS WITH MEANING
18 Months	8-10 WORDS VOCABULARY
2 years	2-3 WORD SENTENCES, USED PRONOUNS, “I”,“ME”, “YOU”
3 years	ASKS QUESTIONS; KNOWS FULL NAME AND GENDER
4 years	SAYS SONG OR POEM; TELLS STORIES
5 years	ASKS MEANING OF WORDS

Infant fixates on her mother as she talks to her at 1 month

Grasping 'with the eye' at 3 months



Diagonal localization of the source of sound at 10 months

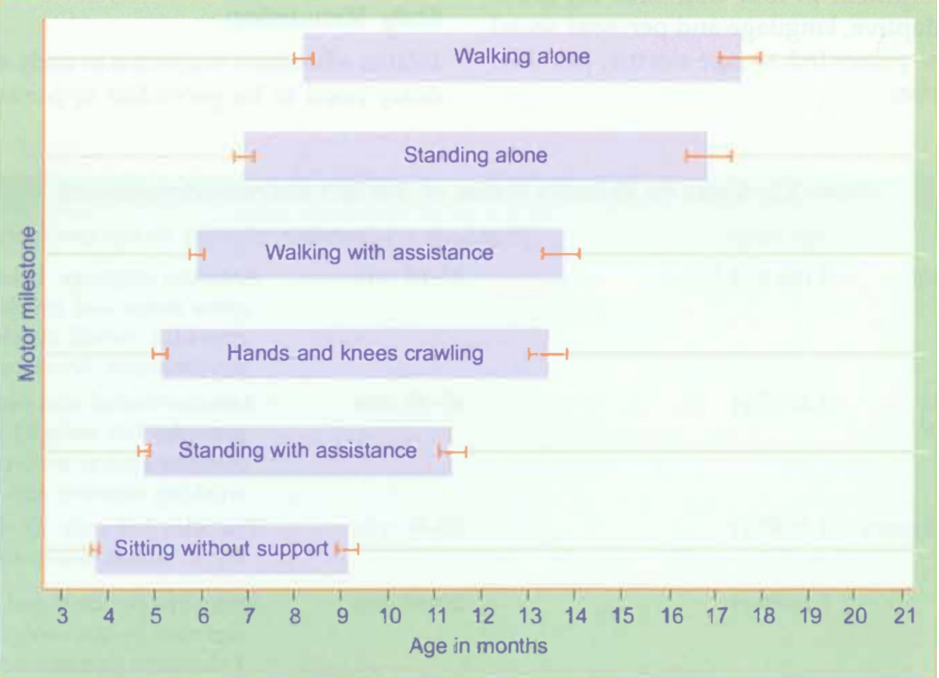
DEVELOPMENTAL DELAY

DEVELOPMENTAL QUOTIENT (DQ)=
DEVELOPMENTAL AGE/ CHRONOLOGICAL AGE X 100

IQ= mental age/ chronological age x 100

DQ<70% -> **DEVELOPMENTAL DELAY**

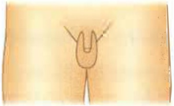
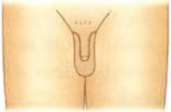
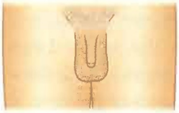
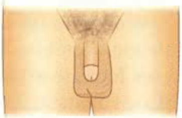
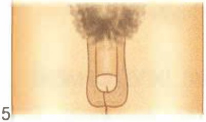
If it involves two or more field -> **GLOBAL DEVELOPMENTAL DELAY** Eg. cerebral palsy



Windows of achievement of six major motor milestones

SEXUAL DEVELOPMENT IN CHILD

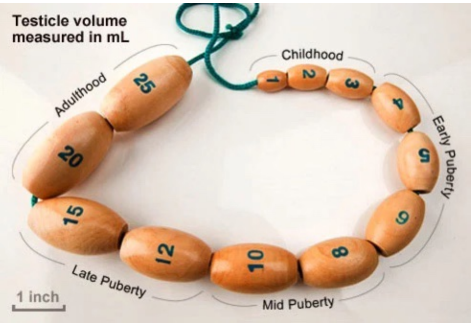
MALES :

1 	< 3 ml < 2.5 cm	Prepubertal
2 	4 ml 2.5–3.2 cm	Reddening of scrotum, testicular volume reaching 4 ml or length 2.5 cm Scanty hair at penile base
3 	10 ml 3.3–4.0 cm	Increase in length of penile shaft Further increase in testicular volume Hair begin to curl and darken
4 	16 ml 4.1–4.5 cm	Increase in girth of penis and glans Darkening of scrotum Coarse, abundant and curly hair, less than in adult
5 	25 ml >4.5 cm	Adult size scrotum and penis Adult type hair, spreading over to medial thighs

Sexual maturity rating (1-5) in boys

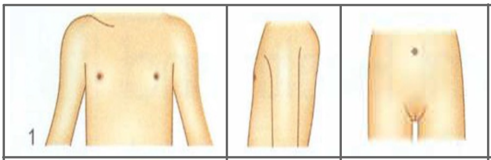
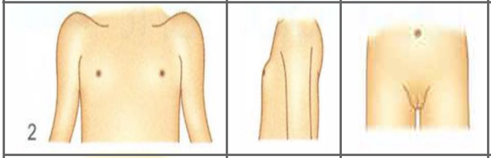
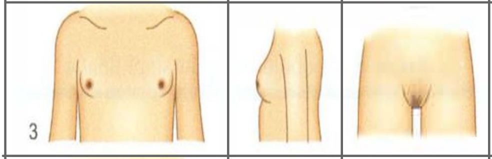
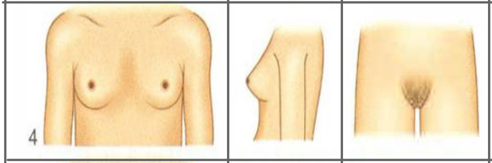
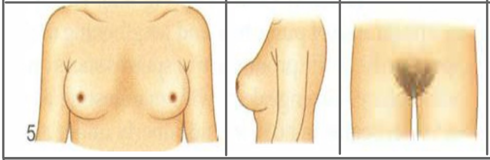
Testicular enlargement
↓
Penile enlargement
↓
Pubic hair
↓
Growth spurt
↓
Axillary hair
↓
Facial hair

Deepening or ripening of voice


Testicle volume measured in mL
Childhood
1
2
3
4
5
6
8
10
12
15
20
25
Early Puberty
Mid Puberty
Late Puberty
Adulthood
1 inch

First sign of puberty : Testicular enlargement

FEMALES :

	Prepubertal; no terminal hair
	Appearance of breast bud Sparse straight hair along the labia
	Generalised breast enlargement (extending beyond the areola) Pigmented pubic hair, coarse, begin to curl
	Nipple and areola form a second mound over the breast Hair increase in amount, spread over entire mons
	Mature adult type breast; nipple projects and areola recedes Adult type pubic hair in triangle shaped area, spreading over to medial thighs

Sexual maturity rating (1-5) in girls

2 ~ 2.5 years

Growth
↓
Thelarche (Breast development) – first sign
↓
Puberache (Development of pubic and axillary hair)
↓
Growth spurt (Peak increase in growth velocity) – 9 cm/ day
↓
Menarche (Beginning of menstrual period)

First sign of puberty : Growth spurt
First visible sign of puberty : Thelarche (as early as 8 ~ 9 yrs)

Vaccination

TYPES OF VACCINES

Live vaccines	Live Attenuated vaccines	Killed Inactivated vaccines	Toxoids	Cellular fraction vaccines	Recombinant vaccines
•Small pox variola vaccine	•BCG •Typhoid oral •Plague •Oral polio •Yellow fever •Measles •Mumps •Rubella •Intranasal Influenza •Typhus	•Typhoid •Cholera •Pertussis •Plague •Rabies •Salk polio •Intra-muscular influenza •Japanise encephalitis	•Diphtheria •Tetanus	•Meningococcal polysaccharide vaccine •Pneumococcal polysaccharide vaccine •Hepatitis B polypeptide vaccine	•Hepatitis B vaccine

THERMO SENSITIVITY OF VACCINE

Vaccines sensitive to heat

Most ↓ Least

- **BCG** (after reconstitution)
- **OPV**
- **Measles**
- **DPT**
- **BCG** (before reconstitution)
- **DT, TT, Hep.B, JE**

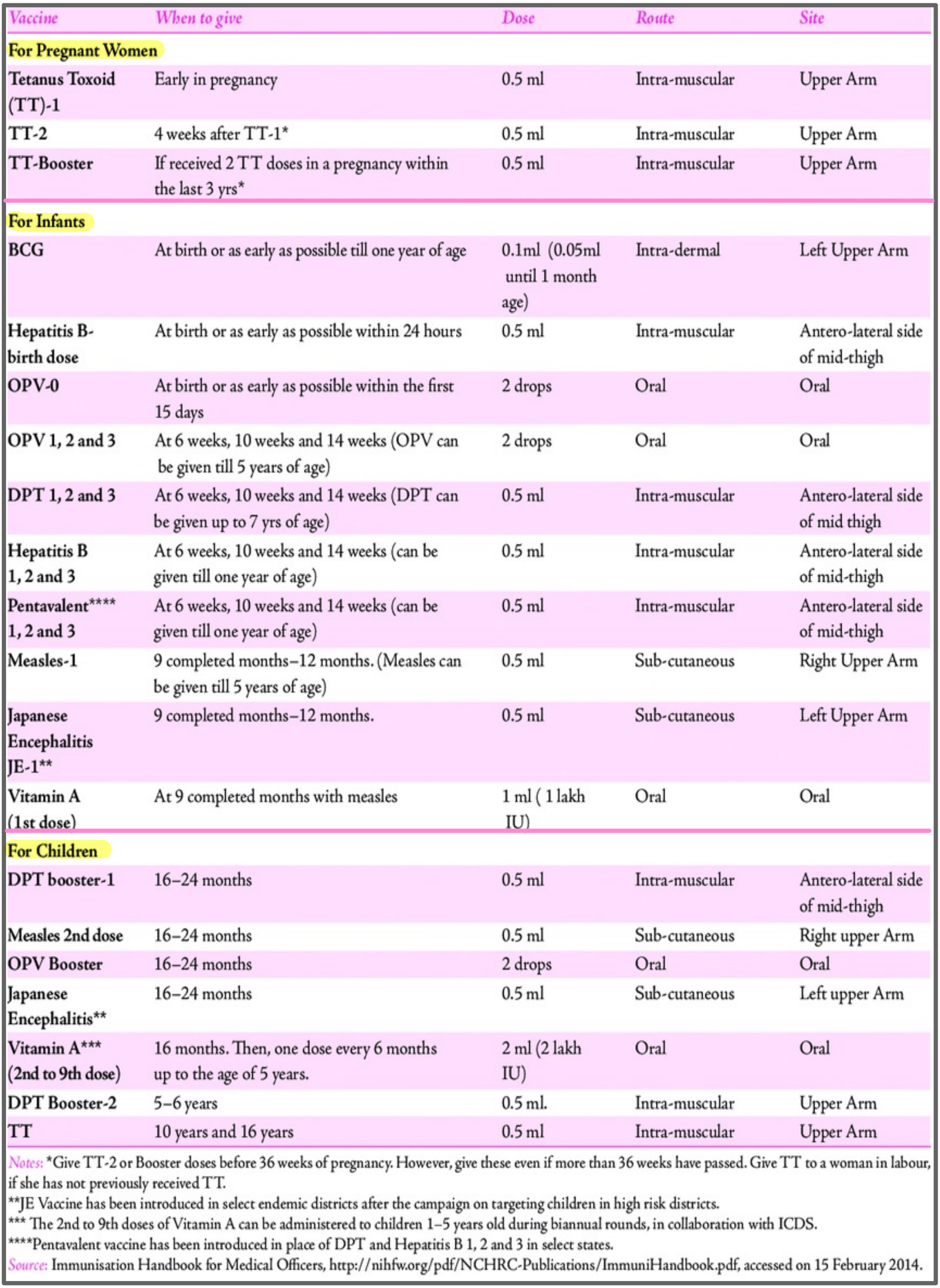
Vaccines sensitive to freezing

Most ↓ Least

- **Hep- B**
- **DPT**
- **DT**
- **TT**

Reference: Essential Pediatrics Ghai (Pg 184)

NATIONAL IMMUNIZATION SCHEDULE



Vaccine	*When to give*	*Dose*	*Route*	*Site*
For Pregnant Women				
Tetanus Toxoid (TT)-1	Early in pregnancy	0.5 ml	Intra-muscular	Upper Arm
TT-2	4 weeks after TT-1*	0.5 ml	Intra-muscular	Upper Arm
TT-Booster	If received 2 TT doses in a pregnancy within the last 3 yrs*	0.5 ml	Intra-muscular	Upper Arm
For Infants				
BCG	At birth or as early as possible till one year of age	0.1ml (0.05ml until 1 month age)	Intra-dermal	Left Upper Arm
Hepatitis B-birth dose	At birth or as early as possible within 24 hours	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
OPV-0	At birth or as early as possible within the first 15 days	2 drops	Oral	Oral
OPV 1, 2 and 3	At 6 weeks, 10 weeks and 14 weeks (OPV can be given till 5 years of age)	2 drops	Oral	Oral
DPT 1, 2 and 3	At 6 weeks, 10 weeks and 14 weeks (DPT can be given up to 7 yrs of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid thigh
Hepatitis B 1, 2 and 3	At 6 weeks, 10 weeks and 14 weeks (can be given till one year of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Pentavalent** 1, 2 and 3**	At 6 weeks, 10 weeks and 14 weeks (can be given till one year of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Measles-1	9 completed months–12 months. (Measles can be given till 5 years of age)	0.5 ml	Sub-cutaneous	Right Upper Arm
Japanese Encephalitis JE-1**	9 completed months–12 months.	0.5 ml	Sub-cutaneous	Left Upper Arm
Vitamin A (1st dose)	At 9 completed months with measles	1 ml (1 lakh IU)	Oral	Oral
For Children				
DPT booster-1	16–24 months	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Measles 2nd dose	16–24 months	0.5 ml	Sub-cutaneous	Right upper Arm
OPV Booster	16–24 months	2 drops	Oral	Oral
Japanese Encephalitis**	16–24 months	0.5 ml	Sub-cutaneous	Left upper Arm
Vitamin A* (2nd to 9th dose)**	16 months. Then, one dose every 6 months up to the age of 5 years.	2 ml (2 lakh IU)	Oral	Oral
DPT Booster-2	5–6 years	0.5 ml.	Intra-muscular	Upper Arm
TT	10 years and 16 years	0.5 ml	Intra-muscular	Upper Arm

Notes: *Give TT-2 or Booster doses before 36 weeks of pregnancy. However, give these even if more than 36 weeks have passed. Give TT to a woman in labour, if she has not previously received TT.

**JE Vaccine has been introduced in select endemic districts after the campaign on targeting children in high risk districts.

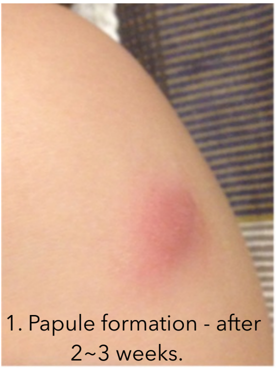
*** The 2nd to 9th doses of Vitamin A can be administered to children 1–5 years old during biannual rounds, in collaboration with ICDS.

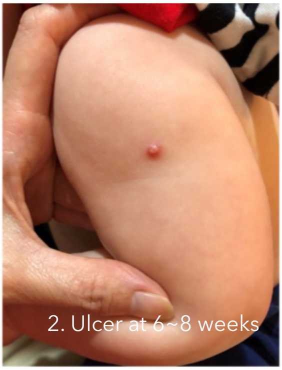
****Pentavalent vaccine has been introduced in place of DPT and Hepatitis B 1, 2 and 3 in select states.

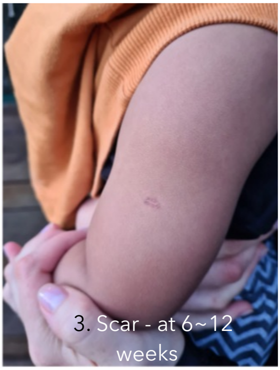
Source: Immunisation Handbook for Medical Officers, http://nihfw.org/pdf/NCHRC-Publications/ImmuniHandbook.pdf, accessed on 15 February 2014.

BACILLUS CALMETTE GUERIN (BCG) VACCINE

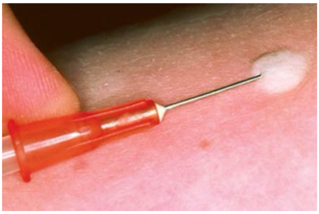
DOSE, ROUTE	0.1 ML; INTRADERMAL
SITE	LEFT UPPER ARM AT INSERTION OF DELTOID
NATIONAL PROGRAM	AT BIRTH; UP TO 1 YR IF MISSED (CATCH UP)
IAP 2012	AS ABOVE;CATCH UP TILL 5 YR
ADVERSE REACTIONS	LOCAL ULCERATION OR DISCHARGING SINUS; AXILLARY LYMPHADENITIS; DISSEMINATED INFECTION, OSTEOMYELTIS OR SCROFULODERMA (IN IMMUNODEFICIENT RECIPIENT)
CONTRAINDICATION	CELLULAR IMMUNODEFICIENCY; SYMPTOMATIC HIV
STORAGE	2-8°C; SENSITIVE TO HEAT AND LIGHT; DISCARD UNUSED VACCINE AFTER 4 HR


1. Papule formation - after 2~3 weeks.


2. Ulcer at 6~8 weeks


3. Scar - at 6~12 weeks

10 Doses
BCG VACCINE
(FREEZE-DRIED)
PASTEUR MÉRIEUX CONNAUGHT



BCG vaccine provides protection against CNS and disseminated TB. (Duration : 20 years)

ORAL POLIOVIRUS (OPV) VACCINE

DOSE, ROUTE	TWO DROPS; ORAL
SCHEDULE NATIONAL PROGRAM	ADMINISTER ONE DOSE AT BIRTH OR WITHIN 15 DAYS (ZERO DOSE); THREE DOSES AT 6, 10 AND 14 WEEKS (PRIMARY) AND TWO DOSES AT 15-18 MO AND 5 YR (BOOSTERS); GIVE ADDITIONAL DOSES ON NIDS AND SIAS; CATCH UP TILL 5 YR
IAP 2012	IN SEQUENTIAL IPV-OPV SCHEDULE: OPV GIVEN AT BIRTH, 6 MO, 9 MO AND 5 MONTHS IF IPV NOT AVAILABLE OR NOT AFFORDED: GIVE OPV AS IN NATIONAL PROGRAM
ADVERSE REACTIONS	VACCINE DERIVED POLIOVIRUS; VACCINE ASSOCIATED PARALYTIC POLIOMYELTIS
CONTRAINDICATIONS	INHERITED OR ACQUIRED IMMUNODEFICIENCY; SYMPTOMATIC HIV
STORAGE	2-8°C; SENSITIVE TO HEAT; USE VACCINE VIAL MONITOR

INACTIVATED POLIOVIRUS (IPV) VACCINE

DOSE, ROUTE	0.5 ML; INTRAMUSCULAR OR SUBCUTANEOUS
NATIONAL PROGRAM	NOT RECOMMENDED
IAP 2012	SEQUENTIAL IPV-OPV SCHEDULE: ADMINISTER 3 DOSES OF IPV AT 6,10 AND14 WEEKS OR 2 DOSES AT 8 AND 16 WEEKS (PRIMARY) AND ONE DOSE AT 15-18 MO (BOOSTER); ALSO GIVE OPV AT BIRTH, 6 MO, 9 MO AND 5 YR
CATCH UP	UP TO 5 YR; 3 DOSES AT 0, 2 AND 6 MO
ADVERSE REACTIONS	LOCAL PAIN, SWELLING
CONTRAINDICATION	KNOWN ALLERGY
STORAGE	2-8°C; SENSITIVE TO LIGHT

DTwP/ DTaP

DOSE, ROUTE	0.5 ML; INTRAMUSCULAR
SITE	ANTEROLATERAL ASPECT OF MID-THIGH (AVOID GLUTEAL REGION: RISK OF SCIATIC NERVE INJURY; INADEQUATE RESPONSE)
NATIONAL PROGRAM	DTwP AT 6, 10 AND 14 WEEKS (PRIMARY); AT 15-18 MO AND 5, 10, 16 YR (BOOSTERS)
IAP 2012	DTaP or DTwP; PRIMARY SCHEDULE AS ABOVE; Tdap/Td AT 10-12 YR; Td EVERY 10YR
Catch up < 7 yr	DTaP or DTwP AT 0, 1 AND 6 MONTHS
Catch up > 7 yr	Tdap at 0 months; Td at 1 and 6 months
ADVERSE REACTIONS	LOCAL PAIN, SWELLING, FEVER (DTwP>DTaP)

MEASLES VACCINE

DOSE, ROUTE	0.5 ML; SUBCUTANEOUS
SITE	RIGHT UPPER ARM (AT INSERTION OF DELTOID) OR ANTEROLATERAL THIGH
NATIONAL PROGRAM	AT 9 MO (6 MO DURING OUTBREAKS; REVACCINATE 4WEEKS LATER, PREFERABLY AT 12-15 MO AS MMR)
IAP 2012	AT 9 MO; ADMINISTER AT LEAST 2 DOSES OF MEASLES CONTAINING VACCINE 4 WEEKS APART; PREFERABLY AS MMR AT 12-15 MO AND4-6 YR
Catch up <12 mo:	ADMINISTER MEASLES VACCINE
Catch up >12 mo:	ADMINISTER MMR VACCINE
ADVERSE REACTIONS	FEVER, TRANSIENT MACULAR RASH ('MEASLES LIKE' ILLNESS) 5-10 DAYS LATER
CONTRAINDICATIONS	(I) IMMUNOSUPPRESSIVE THERAPY (E.G. ALKYLATING AGENTS, HIGH DOSE CORTICOSTEROIDS); (II) MALIGNANCY; (III) SEVERE IMMUNODEFICIENCY (E.G. ADVANCED HN); (IV) UNTREATED TUBERCULOSIS
STORAGE	2-8°C; SENSITIVE TO HEAT AND LIGHT; USE WITHIN4-6 HRS OF RECONSTITUTION

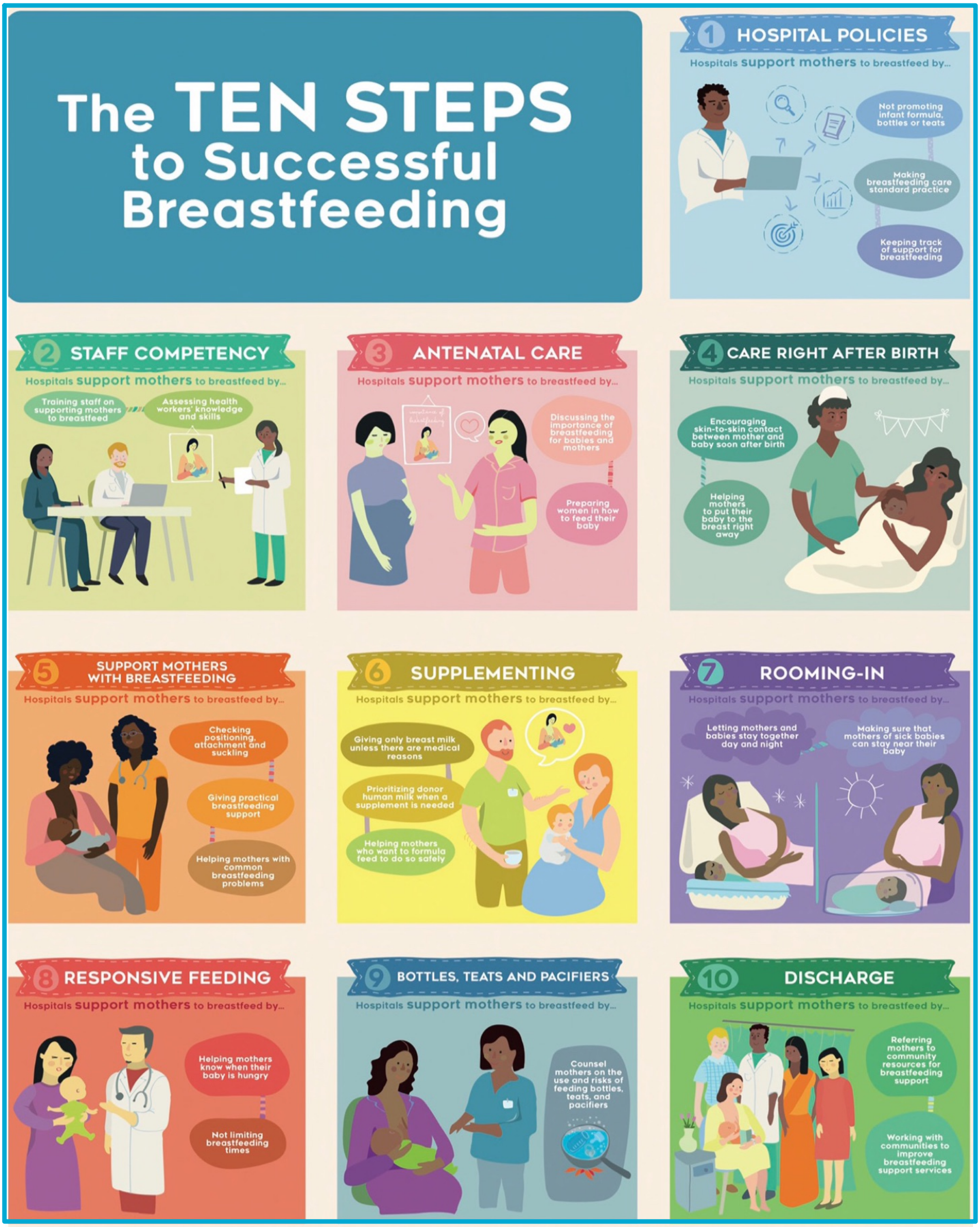
HEPATITIS B VACCINE

DOSE, ROUTE	0.5 ML (1 ML IN ADULTS AND IN CHILDREN RECEIVING HEMODIALYSIS); INTRAMUSCULAR
SITE	ANTEROLATERAL THIGH (DELTOID IN ADULTS); AVOID GLUTEAL REGION
NATIONAL PROGRAM	AT BIRTH (<12-HR-OLD), 6 WEEKS, 14 WEEKS
IAP 2012	AT BIRTH, 6 WEEKS AND 6 MO; MAY GIVE 3-4 DOSES IN AN ALTERNATIVE SCHEDULE* WHILE ENSURING THAT (I) DOSES 1 AND 2 ARE 24 WEEKS APART; (II) DOSES 2 AND 3 ARE 28 WEEKS APART; (III) FINAL DOSE IS AT 26 MO OF AGE AND 216 WEEKS BEYOND FIRST DOSE
Catch up >12 mo	COMPLETE 3 DOSES SERIES; SECOND DOSE IS GIVEN 24 WEEKS AND THIRD DOSE 28 WEEKS AFTER PREVIOUS DOSE
ADVERSE REACTIONS	LOCAL SORENESS; FEVER; FATIGUE
CONTRAINDICATIONS	ANAPHYLAXIS AFTER PREVIOUS DOSE
STORAGE	2-8°C; DO NOT FREEZE

MEASLES, MUMPS, RUBELLA (MMR) VACCINE

DOSE, ROUTE	0.5 ML; SUBCUTANEOUS
SITE	RIGHT UPPER ARM (AT INSERTION OF DELTOID) OR ANTEROLATERAL THIGH
NATIONAL PROGRAM	AT 15-18 MO (ONLY IN SOME STATES)
IAP 2012	TWO DOSES AT 15 (12-18) MO AND 4-6 YR; MINIMUM AGE 12 MO; SECOND DOSE MAY BE GIVEN AT <4 YR BUT 24 WEEKS AFTER THE FIRST DOSE
Catch up >12 mo	ADMINISTER 2 DOSES 24 WEEKS APART; ONE DOSE IF RECEIVED MMR VACCINE PREVIOUSLY
ADVERSE REACTIONS	FEVER, TRANSIENT RASH, ARTHRALGIA, ASEPTIC MENINGITIS, LYMPHADENOPATHY
CONTRAINDICATIONS	(I) IMMUNOSUPPRESSIVE THERAPY (E.G. ALKYLATING AGENTS, HIGH DOSE CORTICOSTEROIDS); (II) MALIGNANCY; (III) SEVERE IMMUNODEFICIENCY (E.G. ADVANCED HN); (IV) UNTREATED TUBERCULOSIS
STORAGE	2-8°C; SENSITIVE TO HEAT AND LIGHT; USE WITHIN 4-6 HR OF RECONSTITUTION

BREAST FEEDING



The TEN STEPS to Successful Breastfeeding

1 HOSPITAL POLICIES

Hospitals support mothers to breastfeed by...

- Not promoting infant formula, bottles or teats
- Making breastfeeding care standard practice
- Keeping track of support for breastfeeding

2 STAFF COMPETENCY

Hospitals support mothers to breastfeed by...

- Training staff on supporting mothers to breastfeed
- Assessing health workers' knowledge and skills

3 ANTENATAL CARE

Hospitals support mothers to breastfeed by...

- Discussing the importance of breastfeeding for babies and mothers
- Preparing women in how to feed their baby

4 CARE RIGHT AFTER BIRTH

Hospitals support mothers to breastfeed by...

- Encouraging skin-to-skin contact between mother and baby soon after birth
- Helping mothers to put their baby to the breast right away

5 SUPPORT MOTHERS WITH BREASTFEEDING

Hospitals support mothers to breastfeed by...

- Checking positioning, attachment and suckling
- Giving practical breastfeeding support
- Helping mothers with common breastfeeding problems

6 SUPPLEMENTING

Hospitals support mothers to breastfeed by...

- Giving only breast milk unless there are medical reasons
- Prioritizing donor human milk when a supplement is needed
- Helping mothers who want to formula feed to do so safely

7 ROOMING-IN

Hospitals support mothers to breastfeed by...

- Letting mothers and babies stay together day and night
- Making sure that mothers of sick babies can stay near their baby

8 RESPONSIVE FEEDING

Hospitals support mothers to breastfeed by...

- Helping mothers know when their baby is hungry
- Not limiting breastfeeding times

9 BOTTLES, TEATS AND PACIFIERS

Hospitals support mothers to breastfeed by...

- Counsel mothers on the use and risks of feeding bottles, teats, and pacifiers

10 DISCHARGE

Hospitals support mothers to breastfeed by...

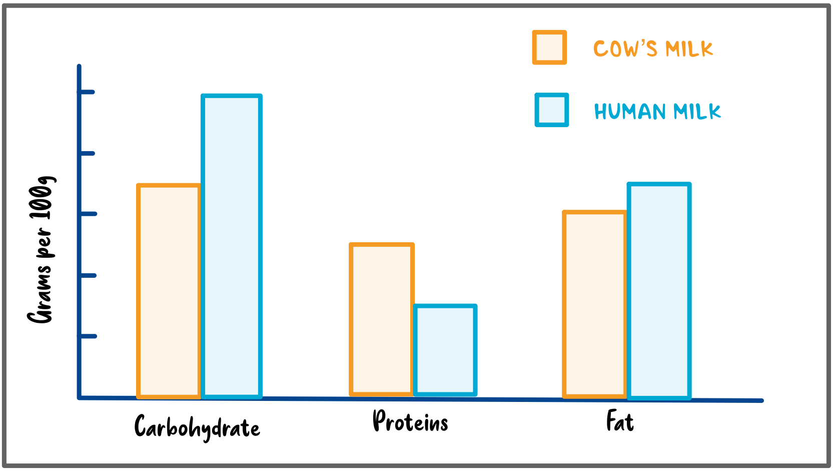
- Referring mothers to community resources for breastfeeding support
- Working with communities to improve breastfeeding support services

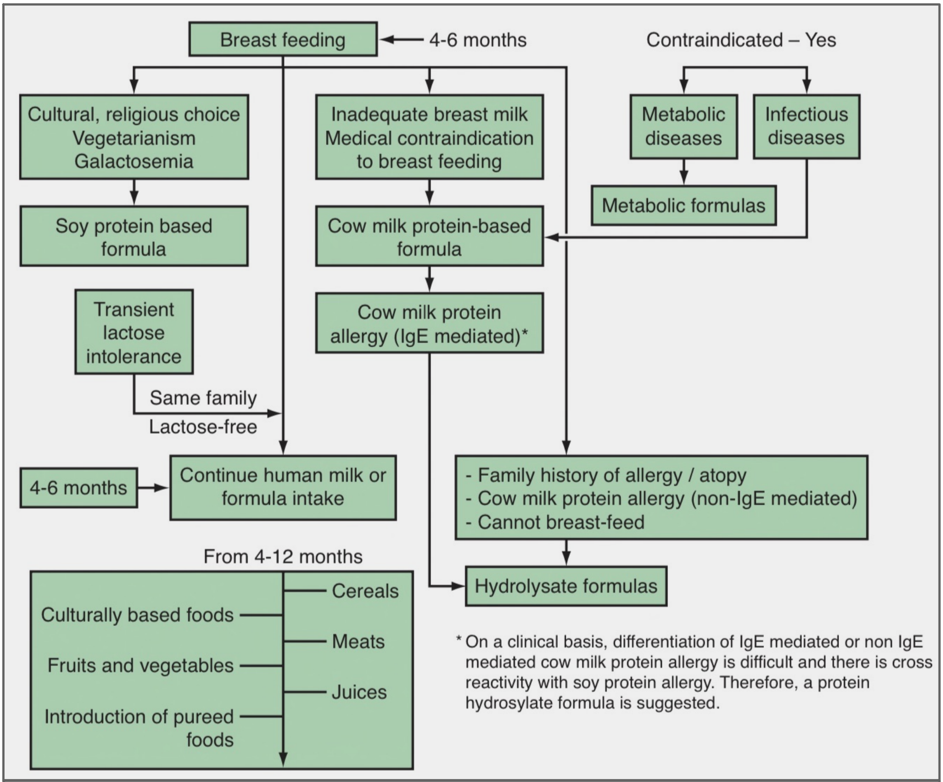
Reference: Essential Pediatrics Ghai (Pg 88)

Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 99)

BREAST MILK

CALORIE CONTENT : - 67 kcal / 100 ml

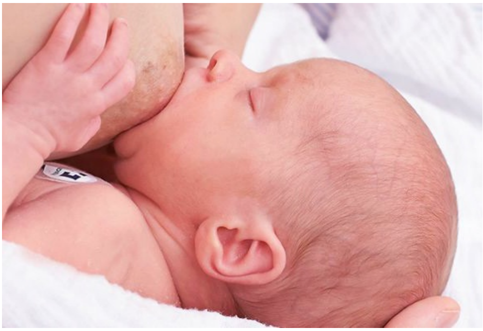



Breast feeding
4-6 months
Contraindicated – Yes
Cultural, religious choice
Vegetarianism
Galactosemia
Inadequate breast milk
Medical contraindication to breast feeding
Metabolic diseases
Infectious diseases
Metabolic formulas
Soy protein based formula
Cow milk protein-based formula
Cow milk protein allergy (IgE mediated)*
Transient lactose intolerance
Same family
Lactose-free
4-6 months
Continue human milk or formula intake
- Family history of allergy / atopy
- Cow milk protein allergy (non-IgE mediated)
- Cannot breast-feed
From 4-12 months
Hydrolysate formulas
Culturally based foods
Fruits and vegetables
Introduction of pureed foods
Cereals
Meats
Juices

* On a clinical basis, differentiation of IgE mediated or non IgE mediated cow milk protein allergy is difficult and there is cross reactivity with soy protein allergy. Therefore, a protein hydrosylate formula is suggested.

Feeding algorithm for term infants

SIGNS OF GOOD ATTACHMENT WHILE BREASTFEEDING



- Neonate's mouth is wide open with most areola inside baby's mouth, lower lip everted and chin toughing the breast.

IMMUNOLOGICAL BENEFITS OF BREAST MILK

COMPONENTS	PROTECTION AGAINST
LYSOZYME	• Escherichia coli • Salmonella • Entamoeba Histolytica
LACTOFERRIN	• Escherichia coli • Salmonella • Shigella • Entamoeba Histolytica • Giardia lambia • Plasmodium Falciparum • Rotavirus
BIFIDUS FACTOR	• Escherichia coli • Salmonella • Shigella
LOW LEVEL OF PARA AMINO BENZOIN ACID	• Plasmodium

MALNUTRITION

WHO CLASSFICATION

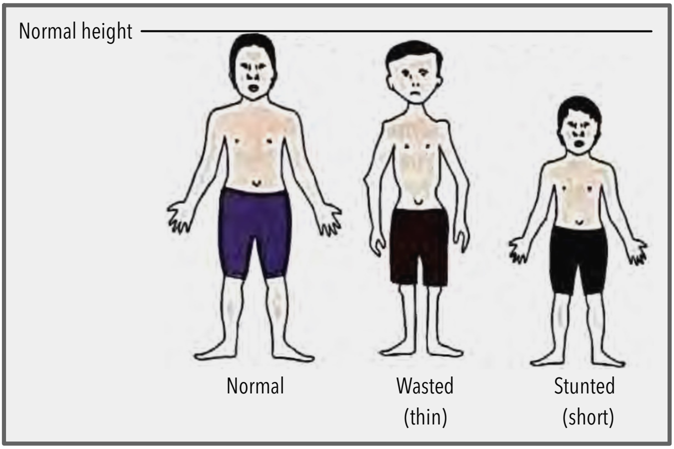
	Moderate malnutrition	Severe malnutrition
Symmetrical oedema	No	Yes (oedematous malnutrition)
Weight-for-height	SD score between -2 to -3	SD score < -3 Severe wasting
Height-for-age	SD score between -2 to -3	SD score < -3 Severe stunting

IAP CLASSIFICATION

Grade of malnutrition	Weight for age of the standard (median) %
Normal	>80
Grade I	71-80 (mild malnutrition)
Grade II	61-70 (moderate malnutrition)
Grade III	51-60 (severe malnutrition)
Grade IV	<50 (very severe malnutrition)

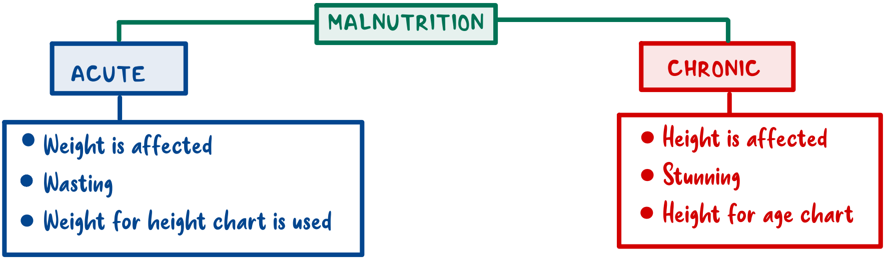
[NEET '19]

Severe acute malnutrition as per who criteria-



Classification of undernutrition

Classification	Criteria	As per WHO growth standards	Sub-classification	As per WHO Reference Standards
Underweight	Low-weight-for-age	Weight-for-age less than minus 2 SD (< -2 SD)	Moderate underweight	Weight-for-age < -2 SD to -3 SD
			Severe underweight	Weight-for-age < -3 SD
Stunting	Low-height (or length) for-age	Height-for-age < -2 SD	Moderate stunting	Height-for-age < -2 SD to -3 SD
			Severe stunting	Height-for-age < -3 SD
Wasting	Low-weight-for-height	Weight-for-height < -2 SD	Moderate wasting	Weight-for-height < -2 SD to -3 SD
			Severe wasting	weight-for-height < -3 SD



MALNUTRITION

ACUTE

- Weight is affected
- Wasting
- Weight for height chart is used

CHRONIC

- Height is affected
- Stunning
- Height for age chart

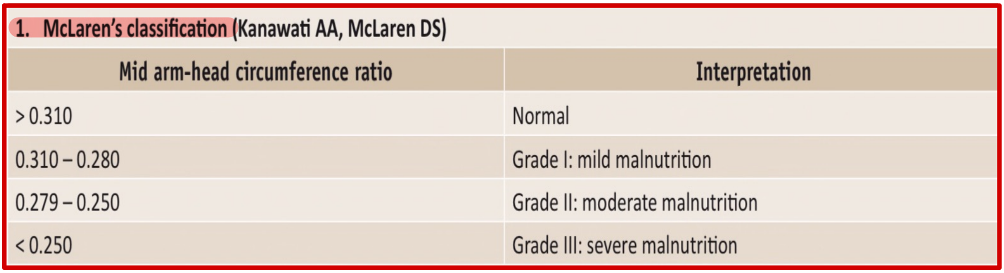
COMPLICATION OF MALNUTRITION

- SUGARS- HYPOGLYCEMIA
- HYPOTHERMIA
- INFECTIONS
- ELECTROLYTE IMBALANCE
- DEHYDRATION
- DEFICIENCES

} → Associated with death

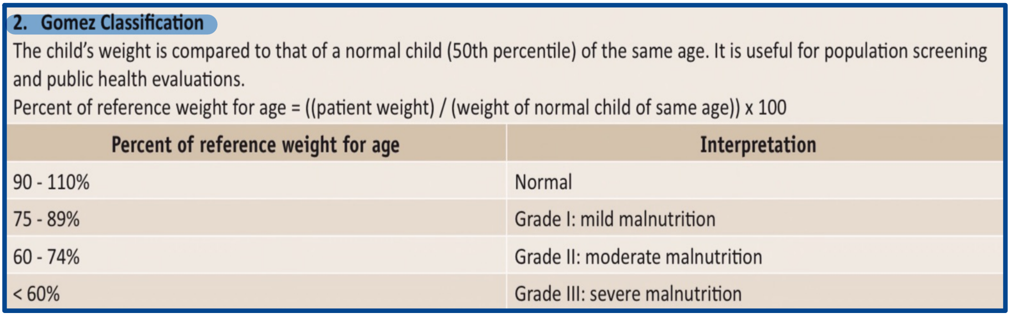
MNEMONIC
SHIELDED

CLASSIFICATION OF MALNUTRITION IN CHILDREN



1. McLaren's classification (Kanawati AA, McLaren DS)

Mid arm-head circumference ratio	Interpretation
> 0.310	Normal
0.310 – 0.280	Grade I: mild malnutrition
0.279 – 0.250	Grade II: moderate malnutrition
< 0.250	Grade III: severe malnutrition

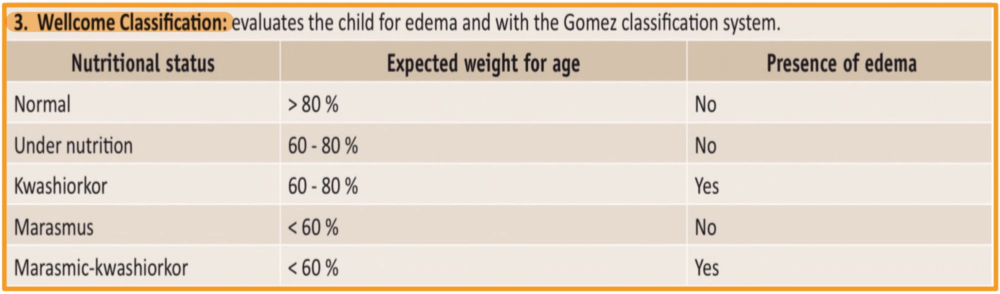


2. Gomez Classification

The child's weight is compared to that of a normal child (50th percentile) of the same age. It is useful for population screening and public health evaluations.

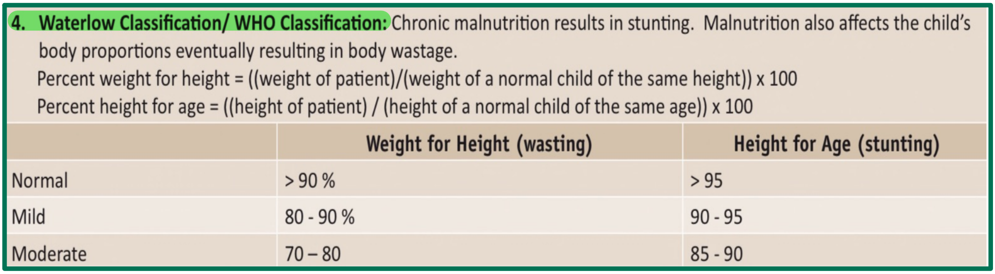
Percent of reference weight for age = ((patient weight) / (weight of normal child of same age)) x 100

Percent of reference weight for age	Interpretation
90 - 110%	Normal
75 - 89%	Grade I: mild malnutrition
60 - 74%	Grade II: moderate malnutrition
< 60%	Grade III: severe malnutrition



3. Wellcome Classification: evaluates the child for edema and with the Gomez classification system.

Nutritional status	Expected weight for age	Presence of edema
Normal	> 80 %	No
Under nutrition	60 - 80 %	No
Kwashiorkor	60 - 80 %	Yes
Marasmus	< 60 %	No
Marasmic-kwashiorkor	< 60 %	Yes



4. Waterlow Classification/ WHO Classification: Chronic malnutrition results in stunting. Malnutrition also affects the child's body proportions eventually resulting in body wastage.

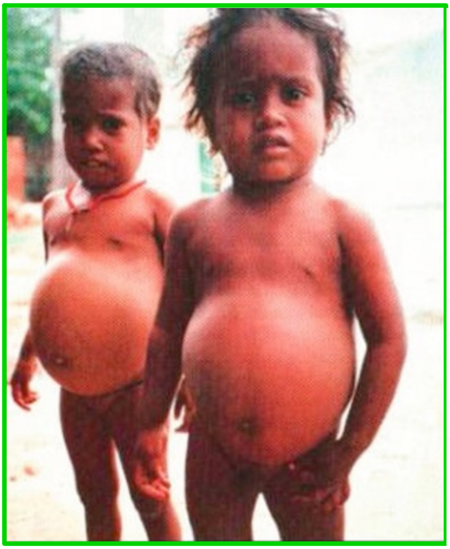
Percent weight for height = ((weight of patient)/(weight of a normal child of the same height)) x 100

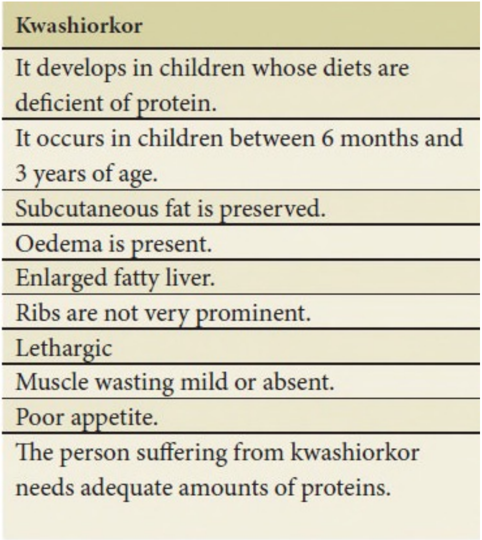
Percent height for age = ((height of patient) / (height of a normal child of the same age)) x 100

	Weight for Height (wasting)	Height for Age (stunting)
Normal	> 90 %	> 95
Mild	80 - 90 %	90 - 95
Moderate	70 – 80	85 - 90

SEVERE ACUTE MALNUTRITION

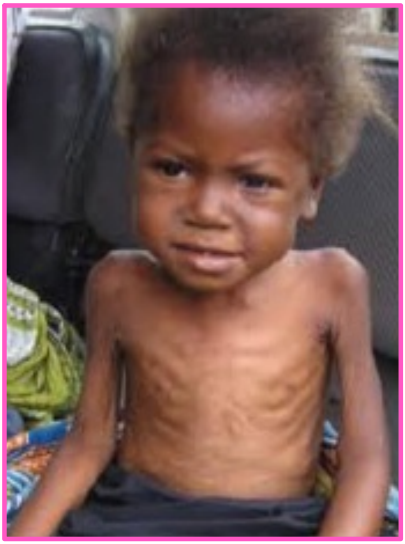
KWASHIORKOR

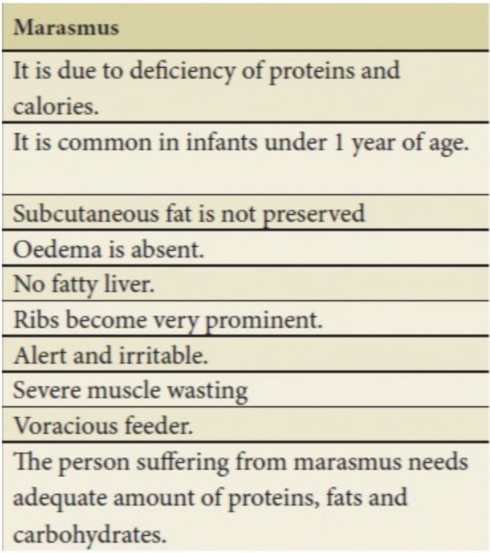




Kwashiorkor
It develops in children whose diets are deficient of protein.
It occurs in children between 6 months and 3 years of age.
Subcutaneous fat is preserved.
Oedema is present.
Enlarged fatty liver.
Ribs are not very prominent.
Lethargic
Muscle wasting mild or absent.
Poor appetite.
The person suffering from kwashiorkor needs adequate amounts of proteins.

MARASMUS



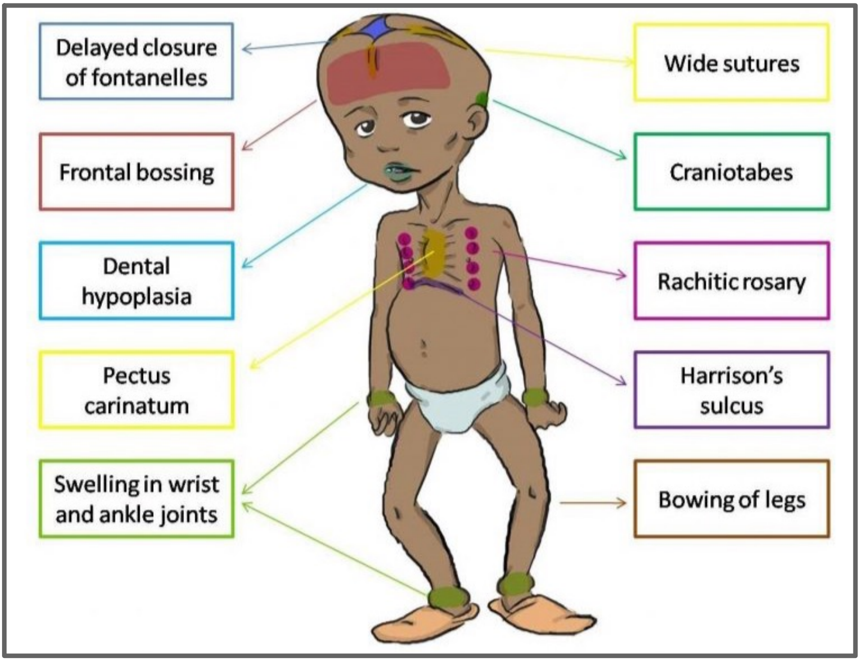


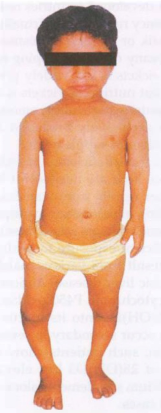
Marasmus
It is due to deficiency of proteins and calories.
It is common in infants under 1 year of age.
Subcutaneous fat is not preserved
Oedema is absent.
No fatty liver.
Ribs become very prominent.
Alert and irritable.
Severe muscle wasting
Voracious feeder.
The person suffering from marasmus needs adequate amount of proteins, fats and carbohydrates.

RICKETS

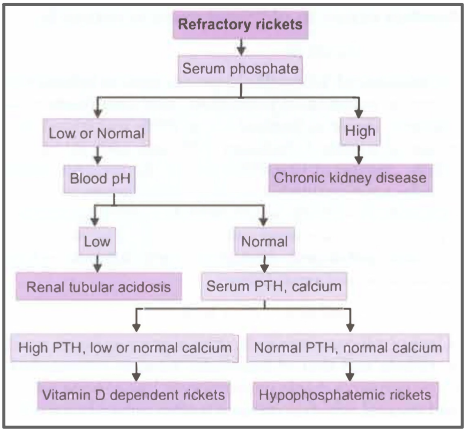
- DEFECTIVE MINERALISATION OF THE GROWING BONE.
- Most common cause - VITAMIN D DEFICIENCY

CLINICAL FEATURES


Delayed closure of fontanelles
Wide sutures
Frontal bossing
Craniotabes
Dental hypoplasia
Rachitic rosary
Pectus carinatum
Harrison's sulcus
Swelling in wrist and ankle joints
Bowing of legs



A 5-yr-old child with rickets with wide wrists and bow legs


Refractory rickets
Serum phosphate
Low or Normal
High
Blood pH
Chronic kidney disease
Low
Normal
Renal tubular acidosis
Serum PTH, calcium
High PTH, low or normal calcium
Normal PTH, normal calcium
Vitamin D dependent rickets
Hypophosphatemic rickets

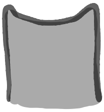
DIAGNOSIS

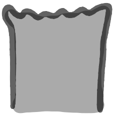
25 OH D is measured - precursor , Decreased in Vitamin D deficiency

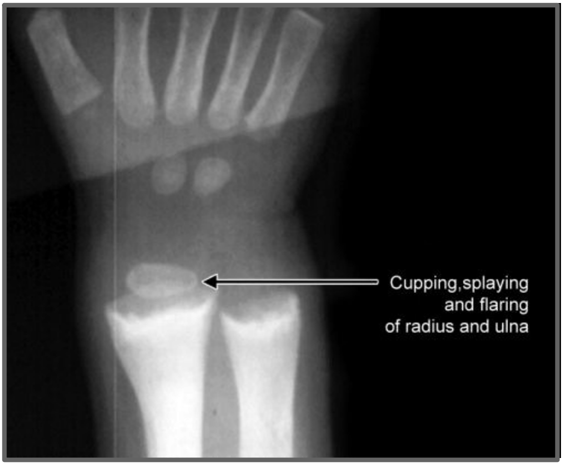
X-ray

- Splaying
- Fraying
- Cupping

} At end of metaphysics (growth plate)


Cupping


Fraying


Cupping,splaying and flaring of radius and ulna

TREATMENT

- Vit D -> 3 to 6 lakh IU given as multiple divided doses
- Available in powders and capsules of 60, 000 IU
- Single massive dose is not preferred now - **STOSS REGIMEN**

Response to treatment :

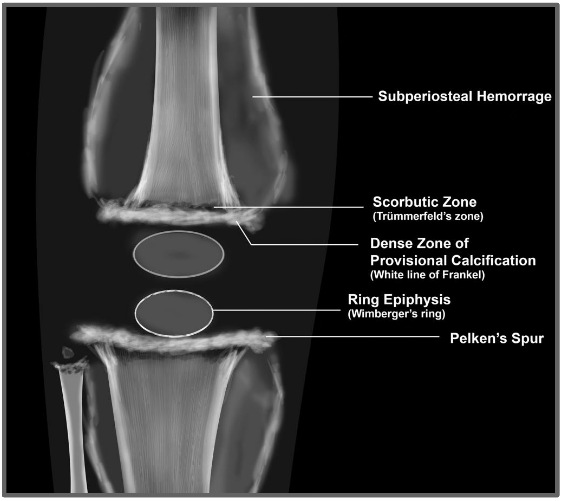
1st response-> repeat X-ray 4 weeks after treatment

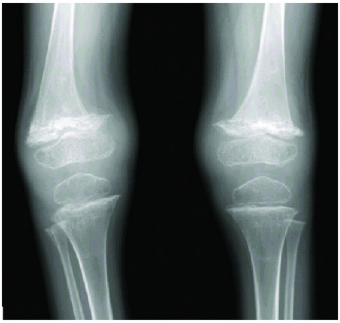
- Thick white line at growth plate -> **HEALING WHITE LINE OF RICKETS**

RADIOLOGICAL SIGNS OF SCURVY

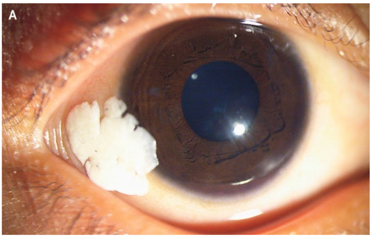
- **WHITE LINE OF FRANKEL**
  - calcified cartilage at metaphysis
- **TRUMMEFELD ZONE**
  - lucent metaphysic area above frankel line
- **WIMBERGER'S RING**
  - circular . opaque shadow at epiphyseal center of ossification
- **PELKEN 'S SPUR**

  Lateral prolongation of white line at the cortical Bone

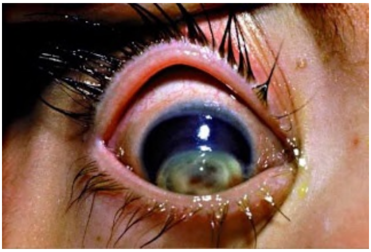

Subperiosteal Hemorrage
Scorbutic Zone
(Trümmerfeld's zone)
Dense Zone of
Provisional Calcification
(White line of Frankel)
Ring Epiphysis
(Wimberger's ring)
Pelken's Spur



VITAMIN A DEFICIENCY

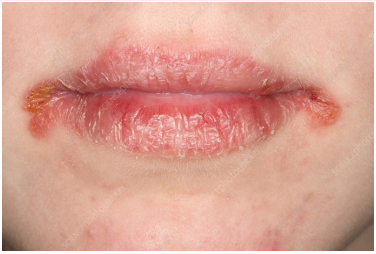


Bitot's spots



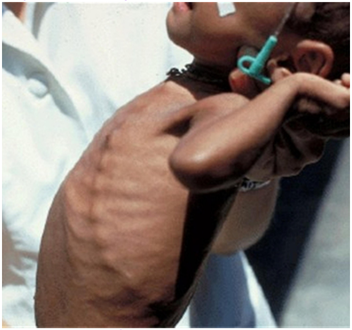
Keratomalacia

VITAMIN B2 DEFICIENCY



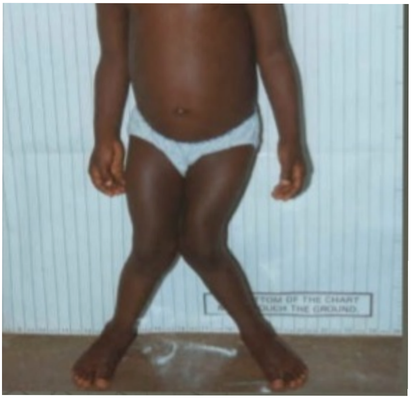
Angular stomatitis and Cheilosis

VITAMIN C DEFICIENCY (SCURVY)

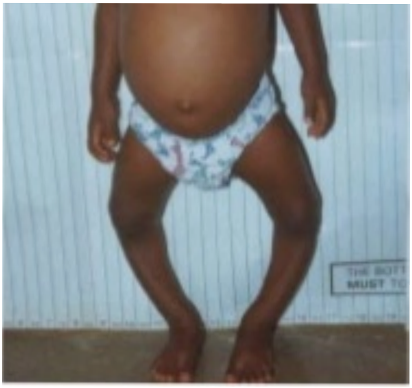


Scorbutic rosary

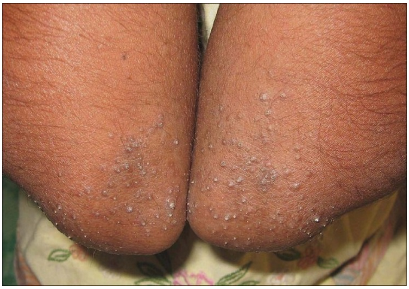
VITAMIN D DEFICIENCY



Knock knee

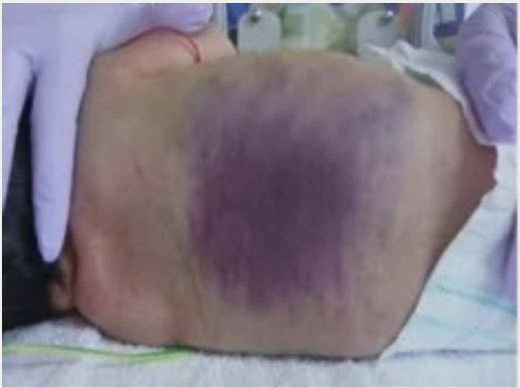


Bow legs

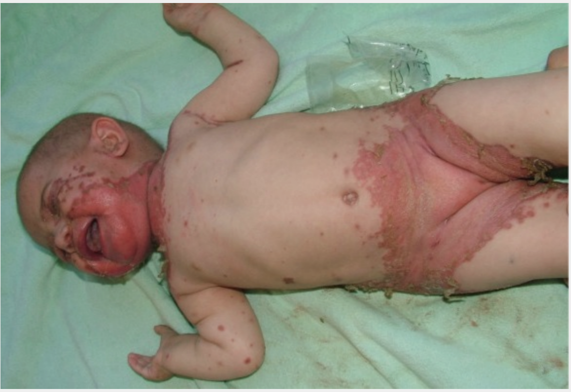


Phrynoderma

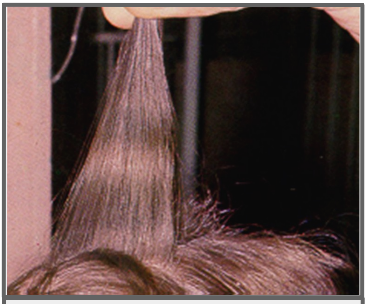
VITAMIN K DEFICIENCY



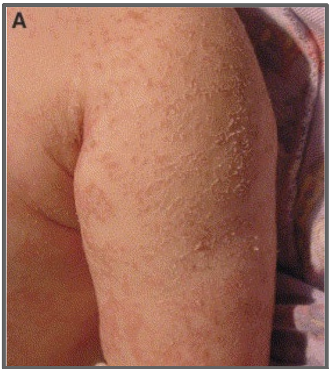
ZINC DEFICIENCY



Acrodermatitis enteropathica



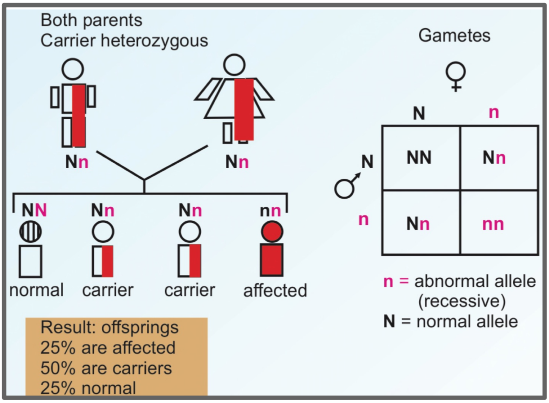
Flag sign



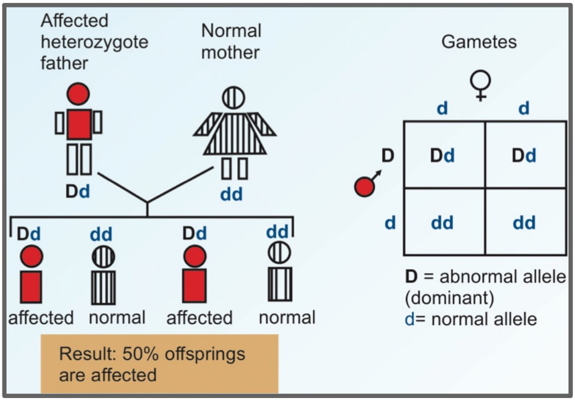
Flaky paint dermatosis

INHERITANCE PATTERN

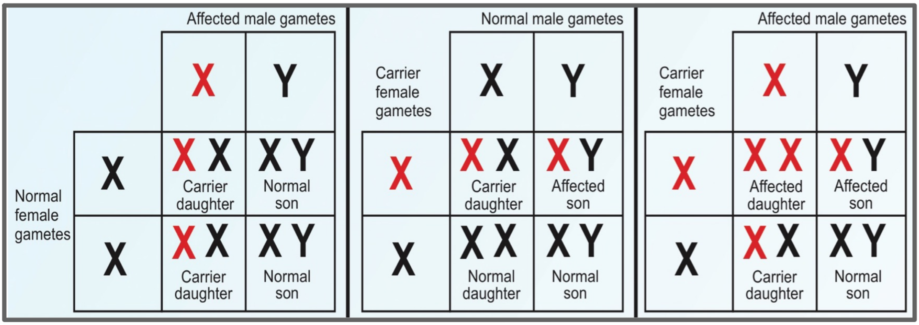
AUTOSOMAL RECESSIVE INHERITANCE


Both parents
Carrier heterozygous
Nn
Nn
NN
Nn
Nn
nn
normal
carrier
carrier
affected
Result: offsprings
25% are affected
50% are carriers
25% normal
Gametes
♀
N
n
♂
N
n
NN
Nn
Nn
nn
n = abnormal allele (recessive)
N = normal allele

AUTOSOMAL DOMINANT INHERITANCE


Affected heterozygote father
Normal mother
Dd
dd
Dd
dd
Dd
dd
affected
normal
affected
normal
Result: 50% offsprings are affected
Gametes
♀
d
d
D
d
Dd
Dd
dd
dd
D = abnormal allele (dominant)
d= normal allele

X-LINKED RECESSIVE INHERITANCE

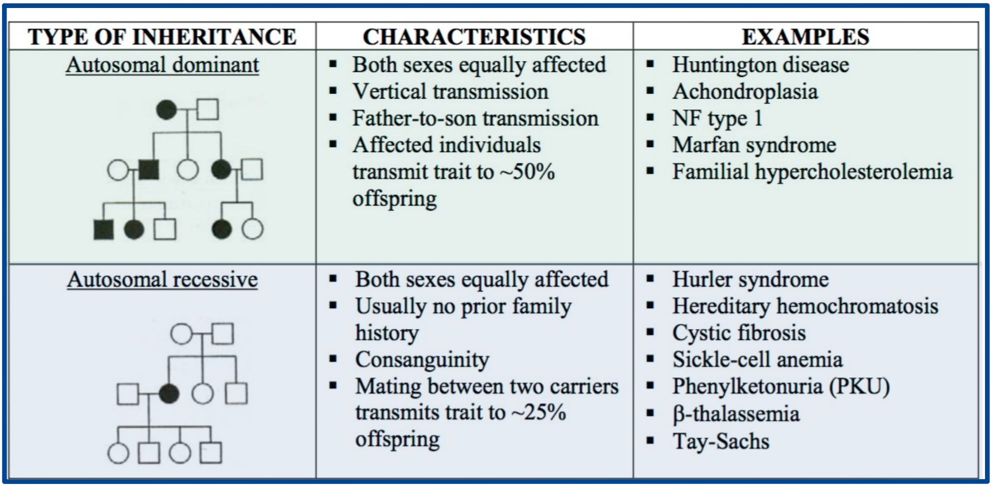


		Affected male gametes	
		X	Y
Normal female gametes	X	XX Carrier daughter	XY Normal son
	X	XX Carrier daughter	XY Normal son

	Normal male gametes	
Carrier female gametes	X	Y
X	XX Carrier daughter	XY Affected son
X	XX Normal daughter	XY Normal son

	Affected male gametes	
Carrier female gametes	X	Y
X	XX Affected daughter	XY Affected son
X	XX Carrier daughter	XY Normal son

Reference: Essential Pediatrics Ghai (Pg 635)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 169)



TYPE OF INHERITANCE	CHARACTERISTICS	EXAMPLES
Autosomal dominant	Both sexes equally affected Vertical transmission Father-to-son transmission Affected individuals transmit trait to ~50% offspring	Huntington disease Achondroplasia NF type 1 Marfan syndrome Familial hypercholesterolemia
Autosomal recessive	Both sexes equally affected Usually no prior family history Consanguinity Mating between two carriers transmits trait to ~25% offspring	Hurler syndrome Hereditary hemochromatosis Cystic fibrosis Sickle-cell anemia Phenylketonuria (PKU) β-thalassemia Tay-Sachs

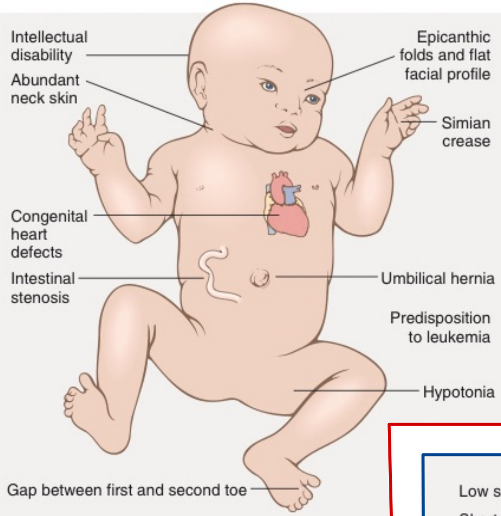
X LINKED RECESSIVE DISEASES

- Red and green colour blindness(Daltonism)
  - mutations of Opsin genes- hybrid genes
- X linked mental retardations
  - Non- specific
  - Fragile X syndrome
- X linked muscular dystrophies (Dystrophin gene)
  - Duchenne muscular dystrophy
  - Becker muscular dystrophy
- X linked coagulation disorders
  - Hemophilia A- deficiency of factor VIII
  - Hemophilia B – deficiency of Factor IX

X LINKED DOMINANT DISEASES

- Vitamin D resistant rickets (hypophosphatemic rickets)

Q. Identify the Type of Inheritance?


Intellectual disability
Abundant neck skin
Epicanthic folds and flat facial profile
Simian crease
Congenital heart defects
Intestinal stenosis
Umbilical hernia
Predisposition to leukemia
Hypotonia
Gap between first and second toe

[NEET '19]

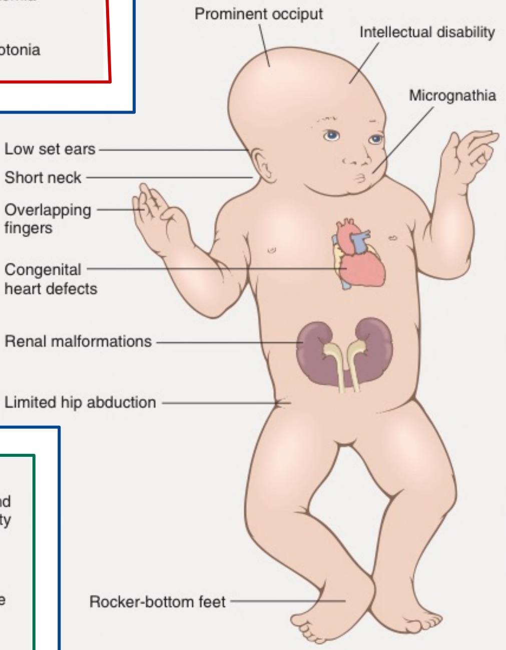
TRISOMY 21: DOWN SYNDROME

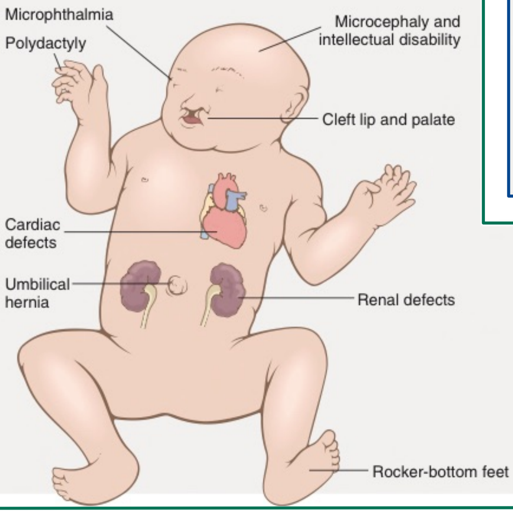
Alzheimer's disease is commonly associated with Down's syndrome.

Incidence: 1 in 700 births
Karyotypes:
Trisomy 21 type: 47,XX, +21
Translocation type: 46,XX,der(14;21)(q10;q10),+21
Mosaic type: 46,XX/47,XX, +21

TRISOMY 18: EDWARDS SYNDROME

Incidence: 1 in 8000 births
Karyotypes:
Trisomy 18 type: 47,XX, +18
Mosaic type: 46,XX/47,XX, +18

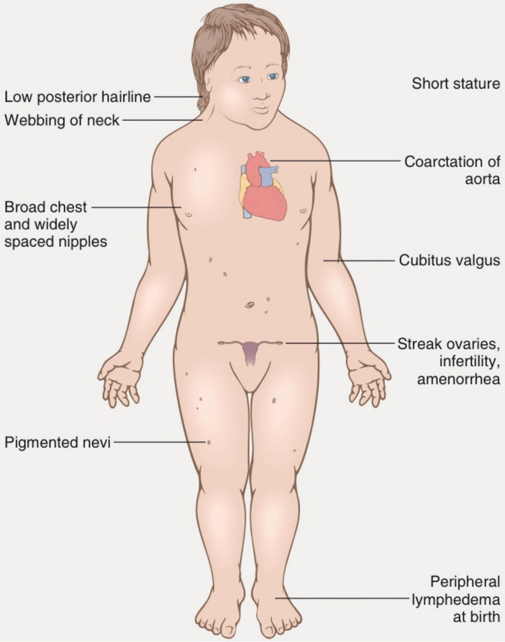

Prominent occiput
Intellectual disability
Micrognathia
Low set ears
Short neck
Overlapping fingers
Congenital heart defects
Renal malformations
Limited hip abduction
Rocker-bottom feet


Microphthalmia
Polydactyly
Microcephaly and intellectual disability
Cleft lip and palate
Cardiac defects
Umbilical hernia
Renal defects
Rocker-bottom feet

TRISOMY 13: PATAU SYNDROME

Incidence: 1 in 15,000 births
Karyotypes:
Trisomy 13 type: 47,XX, +13
Translocation type: 46,XX,+13,der(13;14)(q10;q10)
Mosaic type: 46,XX/47,XX, +13

Robbin & Cotran Pathological basis of diseases (pg 168); Text book of Pathology , Harsh Mohan (pg253)


Low posterior hairline
Webbing of neck
Broad chest and widely spaced nipples
Pigmented nevi
Short stature
Coarctation of aorta
Cubitus valgus
Streak ovaries, infertility, amenorrhea
Peripheral lymphedema at birth

TURNER SYNDROME

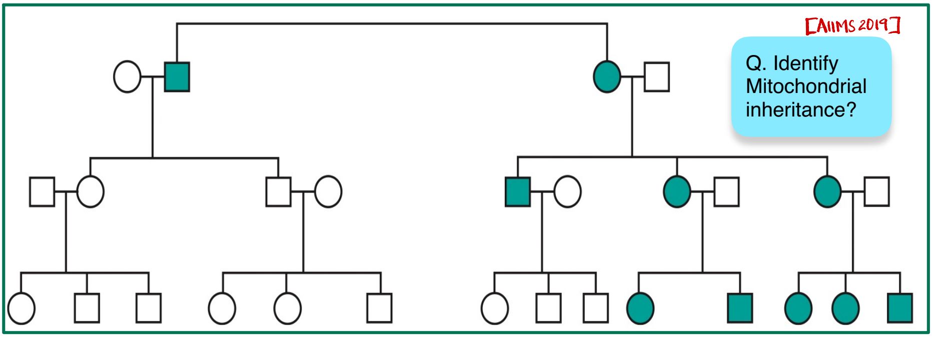
Incidence: 1 in 3000 female births
Karyotypes:
Classic: 45,X
Defective second X chromosome: 46,X,i(Xq)
46,X,del(Xq)
46,X,del(Xp)
46,X, r(X)
Mosaic type: 45,X/46,XX
45,X/46,XY
45,X/47,XXX
45,X/46,X,i(X)(q10)

[AIIMS 2018]

Webbing of neck is associated with higher risk of cardiac anomalies

MITOCHONDRIAL INHERITANCE

- Mutation in the mitochondrial DNA has the characteristic feature of maternal inheritance because the ovum contains the mitochondria with their abundant cytoplasm whereas sperms contains minimal number of mitochondria.
- Mothers transmit their mtDNA to both their sons and daughters, only the daughters are able to transmit the inherited mtDNA to future generations.
- Leber hereditary optic neuropathy is a prototype of this type of disorder



[AIIMS 2019]

Q. Identify Mitochondrial inheritance?

Robbin & Cotran Pathological basis of diseases (pg 168); Text book of Pathology , Harsh Mohan (pg253)

Fluid and Electrolyte Disturbance

BODY WEIGHT (kg)	VOLUME PER DAY	HOURLY RATE
0-10	100 mL/kg	4 mL/kg/hr
11-20	1,000 mL + 50 mL/kg for each 1 kg >10 kg	40 mL/hr + 2 mL/kg/hr × (wt–10)
>20	1,500 mL + 20 mL/kg for each 1 kg >20 kg*	60 mL/hr + 1 mL/kg/hr × (wt–20)†

Body Weight Method for Calculating Maintenance Fluid Volume and Rate

DIARRHOEA

CAUSE OF DEATH - DEHYDRATION

TYPES

- **ACUTE WATERY**
  - Most common
- **DYSENTERY**
  - Blood and mucus in loose stools
- **PERSISTENT DIARRHOEA**
  - > 14 days

	MILD	MODERATE	SEVERE
Infant	5%	10%	15%
Adolescent	3%	6%	9%
Infants and young children	Thirsty, alert; restless	Thirsty; restless or lethargic; irritable	Drowsy; limp, cold, sweaty, cyanotic extremities; may be comatose
Older children	Thirsty, alert	Thirsty, alert (usually)	Usually conscious (but at reduced level), apprehensive; cold, sweaty, cyanotic extremities; wrinkled skin on fingers and toes; muscle cramps
SIGNS AND SYMPTOMS			
Tachycardia	Absent	Present	Present
Palpable pulses	Present	Present (weak)	Decreased
Blood pressure	Normal	Orthostatic hypotension	Hypotension
Cutaneous perfusion	Normal	Normal	Reduced and mottled
Skin turgor	Normal	Slight reduction	Reduced
Fontanelle	Normal	Slightly depressed	Sunken
Mucous membrane	Moist	Dry	Very dry
Tears	Present	Present or absent	Absent
Respirations	Normal	Deep, may be rapid	Deep and rapid
Urine output	Normal	Oliguria	Anuria and severe oliguria

Reference: Essential Pediatrics Ghai (Pg 70)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 125)

DIARRHOEA TREATMENT

NO dehydration	SOME dehydration	SEVERE dehydration
PLAN A	PLAN B	PLAN C
ORS 100 - 200 ml / loose stool (*ongoing losses*)	Rehydration (**75 ml/kg** in first 4 hours) + Maintenance (*Replacing ongoing loss*)	Start IV fluids RL>> NS Give **100 ml/kg** as

Age	30ml/kg	70 ml/kg
< 1yr	1 hr	5 hr
> 1 yr	30 min	2 1/2 hr

Composition	**ReSoMal (mmol/L)**	**Standard ORS (mmol/L)**	**Reduced osmolarity ORS**
Glucose	125	111	75
Sodium	45	90	75
Potassium	40	20	20
Chloride	70	80	65
Citrate	7	10	10
Magnesium	3	...	...
Zinc	0.3	...	...
Copper	0.045	...	...
Osmolarity (mOsm/L)	300	311	245

ReSoMal
FOR REHYDRATION OF PATIENTS WITH SEVERE ACUTE MALNUTRITION
DESTINÉ À LA RÉHYDRATATION DES PATIENTS MALNUTRIS AIGUS SÉVÈRES
1 sachet / 2 litres of water
1 sachet / 2 litres d'eau
Instructions for preparation
Préparation
nutriset

ORAL REHYDRATION SALTS I.P
ELECTRAL
BASED ON W.H.O. FORMULA
NOT FOR INJECTION

NEW
Low
اوسمولار او-آر-ایس
20
SACHETS
Osmolar O.R.S.
Oral Rehydration Salts
Low Osmolarity
WHO recommended NEW O.R.S. Formulation for the restoration of fluid in diarrhoea
ORANGE FLAVOUR
Manufactured by:
The House of Quality Pharmaceuticals
ATCO LABORATORIES LIMITED
B-18, S.I.T.E., KARACHI-PAKISTAN
EHAD MEDICAL CENTRE

CAUSES OF FEVER OF UNKNOWN ORIGIN

INFECTIOUS

Enteric fever, malaria, urinary tract infections, tuberculosis, chronic hepatitis, HIV, occult abscesses (liver, pelvic), mastoiditis, sinusitis, osteomyelitis, meningitis, infectious mononucleosis, infective endocarditis, brucellosis, CMV, toxoplasmosis, kala-azar

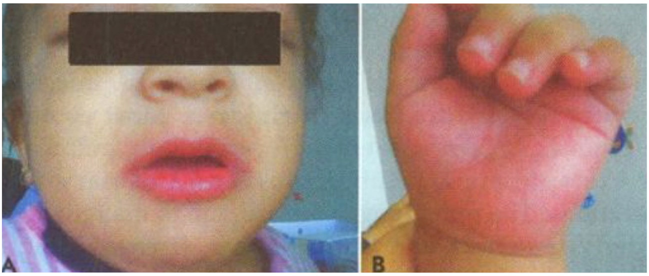
AUTOIMMUNE

Systemic onset juvenile rheumatoid arthritis, Kawasaki disease, systemic lupus erythematosus, inflammatory bowel disease, polyarteritis nodosa

MALIGNANT

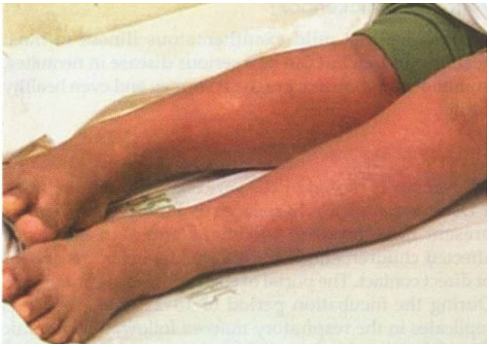
Leukemia, lymphoma, Langerhans cell histiocytosis

KAWASAKI DISEASE:



(A) Red and cracked lips

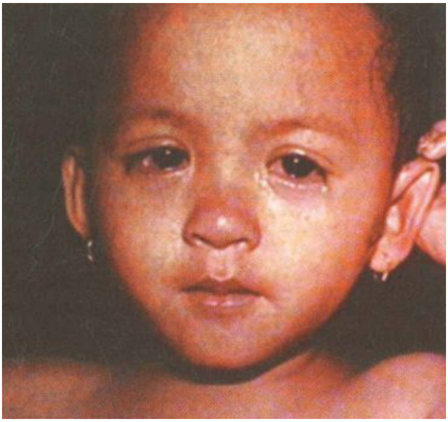
(B) palmar rash and swelling



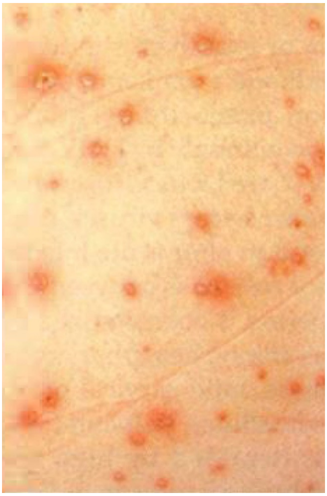
Diffuse erythematous rash seen in a patient with dengue

Reference: Essential Pediatrics Ghai (Pg 209)

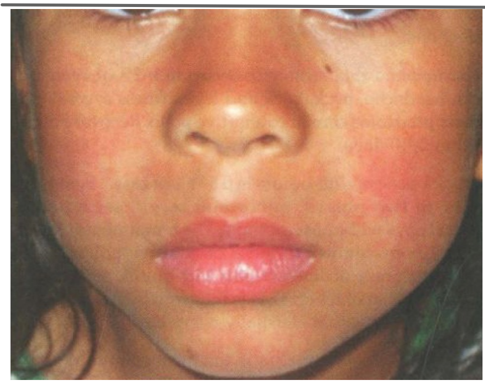
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg361)



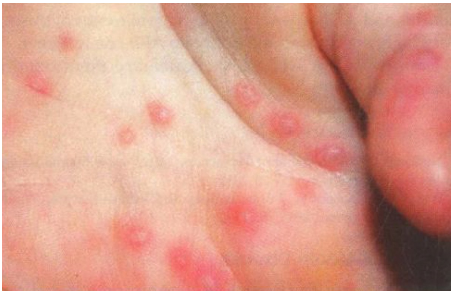
Conjunctival congestion and morbilliform rash in a child with measles



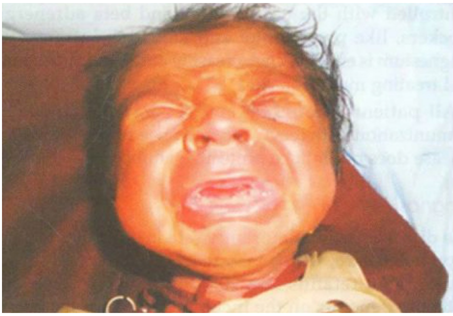
Polymorphic rash of chickenpox



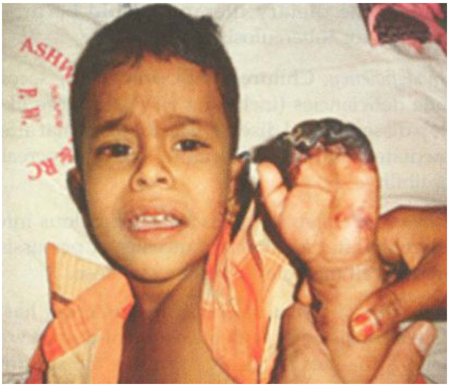
The rash resembles a 'slapped cheek' in erythema infectiosum



vesicular rash of hand-foot-mouth disease



Neonatal tetanus



Necrotic rash in spotted fever

INTRAUTERINE INFECTIONS

T	TOXOPLASMOSIS
O	SYPHILIS, VARICELLA-ZOSTER, PARVOVIRUS B19, ZIKA VIRUS
R	RUBELLA
C	CYTOMEGALOVIRUS
H	HERPES

VARICELLA

Congenital- Intrauterine transmission

- Pregnant female gets chicken pox in Ist trimester- TERATOGENICITY
- Most severe form , limb atrophy/ hypoplasia, cortical atrophy, cicatrise (scars)

Neonatal - Perinatal transmission

- Most common
- Risk period- 5 days before and 2 days after delivery
- Clinical features: Necrotising pneumonia
  Vesicular skin rash

Prevention : VZIG varicella zoster immunoglobin

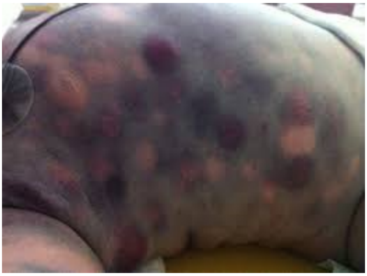
Treatment : ACYCLOVIR TO BOTH MOTHER AND BABY

RUBELLA

- TRIAD
  - CATARACT ,
  - SENSORINEURAL DEAFNESS
  - INVOLVEMENT OF HEART- PDA> PS> VSD> ASD

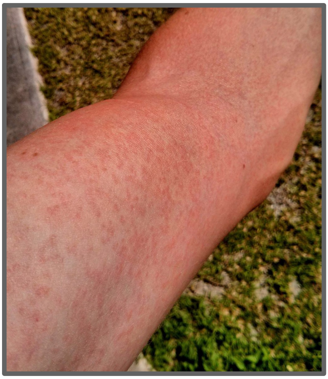
Most common ocular finding - salt and pepper retinopathy

Blue berry muffin - purpurin rash (Also seen in CMV)



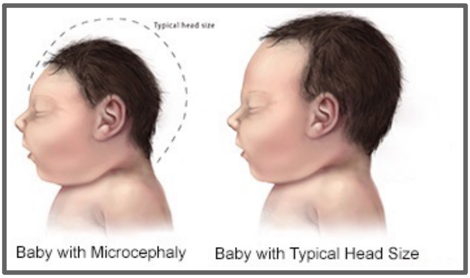
ZIKA FEVER

- Caused by Zika virus (Flaviviridae)
- Spread by Aedes aegypti and A.albopictus



SYMPTOMS

- Fever
- Red eyes
- Joint pains
- Headache
- Maculopapular rash

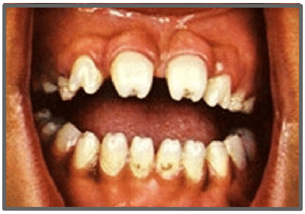


Baby with Microcephaly Baby with Typical Head Size

SYPHILIS

- STD which may cause **FETAL ABNORMALITY**

EARLY	LATE
Rash involving palm and soles Bone involvement - osteochondritis, periosteitis Pseudo paralysis of parrot Rhinitis - snuffles	Hutchinson Triad Mulberry molars Saddle nose deformity Fissures at angles of mouth - **RHAGADES**

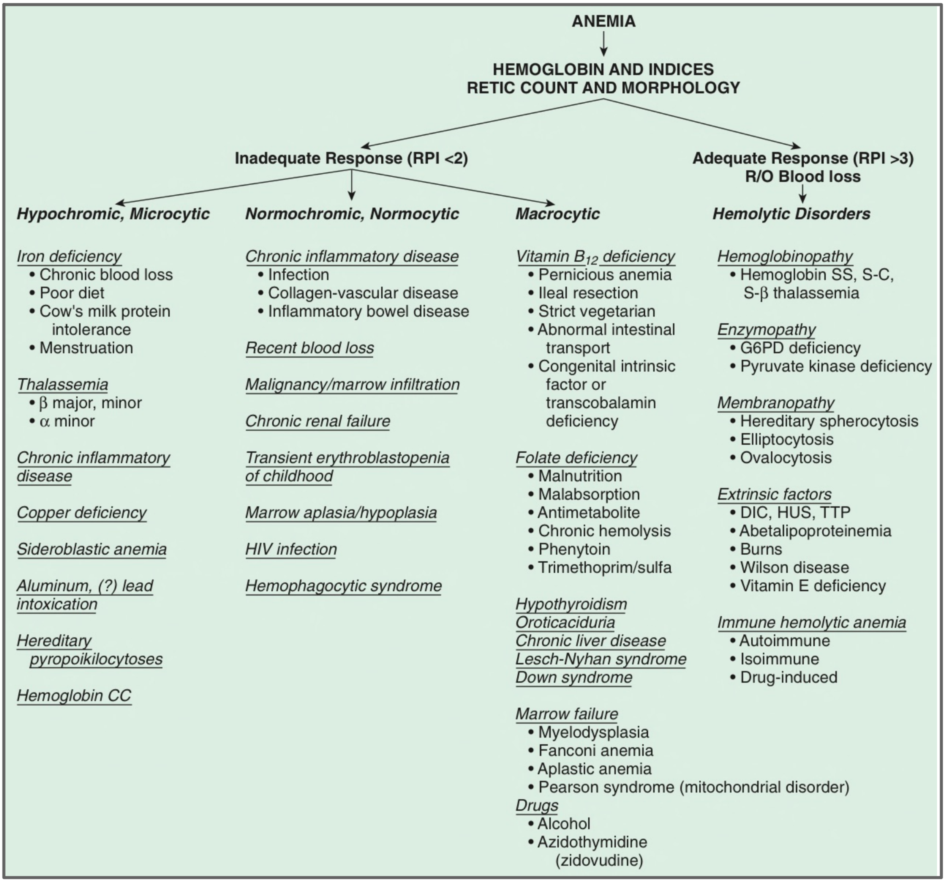


HUTCHINSON TEETH - Notched incisors

HUTCHINSONS TRIAD-
HUTCHINSON TEETH
KERATITIS
DEAFNESS

Q.Saddle nose deformity is seen in?

Haematological Disorders



ANEMIA

↓

HEMOGLOBIN AND INDICES
RETIC COUNT AND MORPHOLOGY

Inadequate Response (RPI <2)

Adequate Response (RPI >3)
R/O Blood loss

Hypochromic, Microcytic

Iron deficiency
- Chronic blood loss
- Poor diet
- Cow's milk protein intolerance
- Menstruation

Thalassemia
- β major, minor
- α minor

Chronic inflammatory disease

Copper deficiency

Sideroblastic anemia

Aluminum, (?) lead intoxication

Hereditary pyropoikilocytoses

Hemoglobin CC

Normochromic, Normocytic

Chronic inflammatory disease
- Infection
- Collagen-vascular disease
- Inflammatory bowel disease

Recent blood loss

Malignancy/marrow infiltration

Chronic renal failure

Transient erythroblastopenia of childhood

Marrow aplasia/hypoplasia

HIV infection

Hemophagocytic syndrome

Macrocytic

Vitamin B_{12} deficiency
- Pernicious anemia
- Ileal resection
- Strict vegetarian
- Abnormal intestinal transport
- Congenital intrinsic factor or transcobalamin deficiency

Folate deficiency
- Malnutrition
- Malabsorption
- Antimetabolite
- Chronic hemolysis
- Phenytoin
- Trimethoprim/sulfa

Hypothyroidism
Oroticaciduria
Chronic liver disease
Lesch-Nyhan syndrome
Down syndrome

Marrow failure
- Myelodysplasia
- Fanconi anemia
- Aplastic anemia
- Pearson syndrome (mitochondrial disorder)

Drugs
- Alcohol
- Azidothymidine (zidovudine)

Hemolytic Disorders

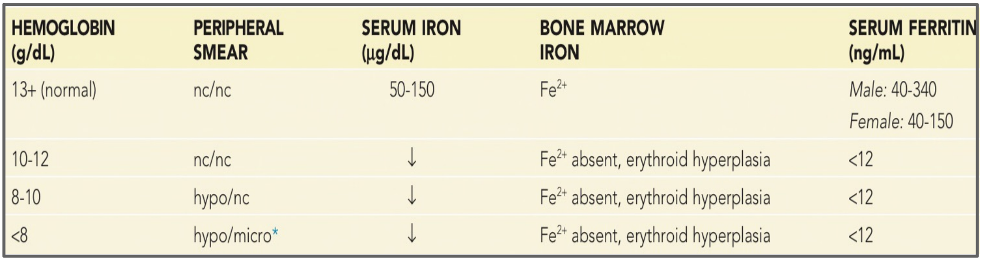
Hemoglobinopathy
- Hemoglobin SS, S-C, S-β thalassemia

Enzymopathy
- G6PD deficiency
- Pyruvate kinase deficiency

Membranopathy
- Hereditary spherocytosis
- Elliptocytosis
- Ovalocytosis

Extrinsic factors
- DIC, HUS, TTP
- Abetalipoproteinemia
- Burns
- Wilson disease
- Vitamin E deficiency

Immune hemolytic anemia
- Autoimmune
- Isoimmune
- Drug-induced

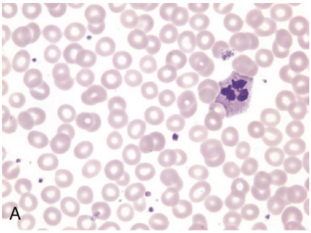


HEMOGLOBIN (g/dL)	PERIPHERAL SMEAR	SERUM IRON (μg/dL)	BONE MARROW IRON	SERUM FERRITIN (ng/mL)
13+ (normal)	nc/nc	50-150	Fe^{2+}	*Male:* 40-340 *Female:* 40-150
10-12	nc/nc	↓	Fe^{2+} absent, erythroid hyperplasia	<12
8-10	hypo/nc	↓	Fe^{2+} absent, erythroid hyperplasia	<12
<8	hypo/micro*	↓	Fe^{2+} absent, erythroid hyperplasia	<12

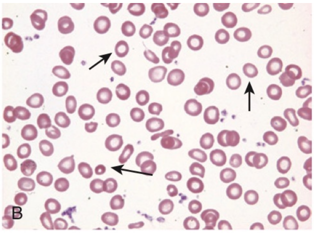
Reference: Essential Pediatrics Ghai (Pg330)

Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 563)

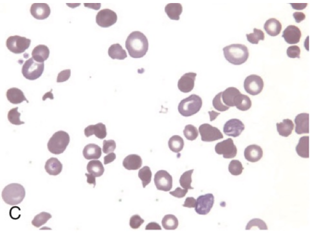
MORPHOLOGICAL ABNORMALITIES OF THE RED BLOOD CELL.



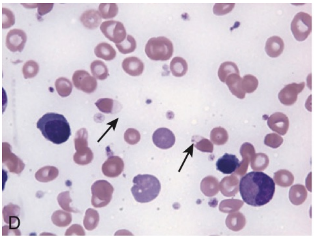
A, Normal.



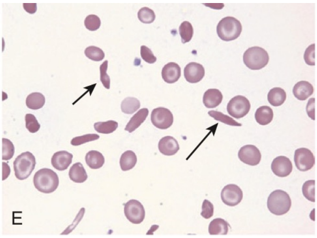
B, Hypochromic microcytes (iron deficiency).



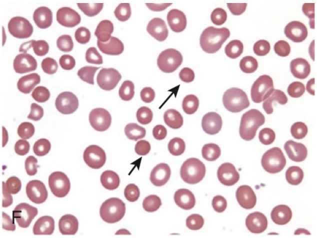
C, Schistocytes (hemolytic uremic syndrome.)



D, Blister cells (glucose-6-phosphate dehydrogenase deficiency).



E, Sickle cells (hemoglobin SS disease).



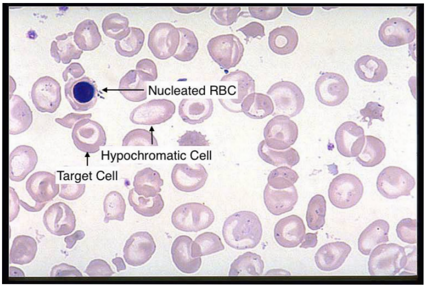
F, Spherocytes (autoimmune hemolytic anemia).

MICROCYTIC ANEMIAS

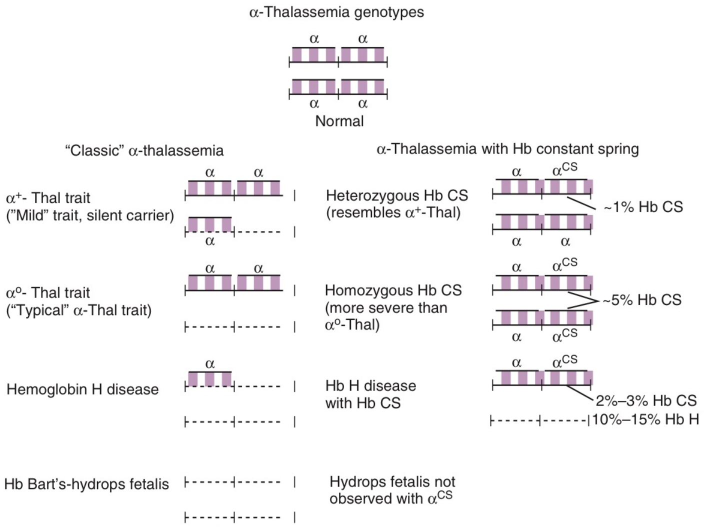
TEST	IRON DEFICIENCY ANEMIA	THALASSEMIA MINOR[†]	ANEMIA OF INFLAMMATION[‡]
Serum iron	Low	Normal	Low
Serum iron-binding capacity	High	Normal	Low or normal
Serum ferritin	Low	Normal or high	Normal or high
Marrow iron stores	Low or absent	Normal or high	Normal or high
Marrow sideroblasts	Decreased or absent	Normal or increased	Normal or increased
Free erythrocyte protoporphyrin	High	Normal or slightly increased	High
Hemoglobin A_2 or F	Normal	High β-thalassemia; normal α-thalassemia	Normal
Red blood cell distribution width[§]	High	Normal	Normal/↑

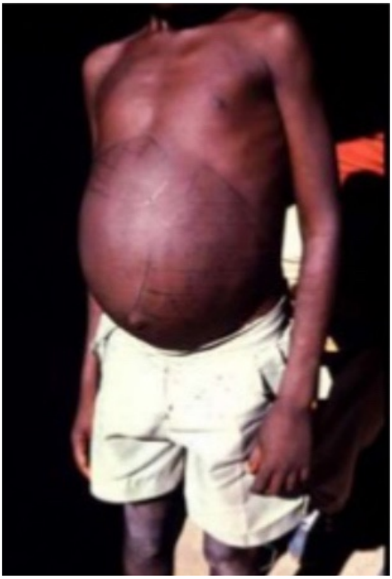
THALASSEMIA

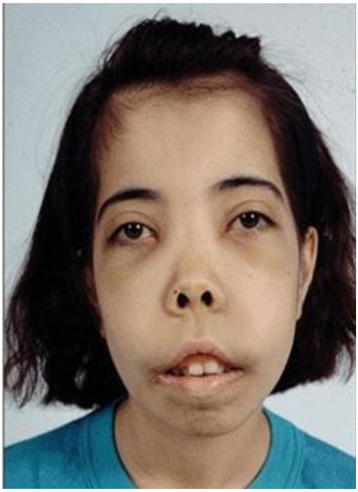
- Thalassemia is a heterogeneous disorder
- Caused by germline mutations that decrease the synthesis of α-globin or β-globin
- Leads to microcytic anemia, tissue hypoxia, and red cell hemolysis


Nucleated RBC
Hypochromatic Cell
Target Cell

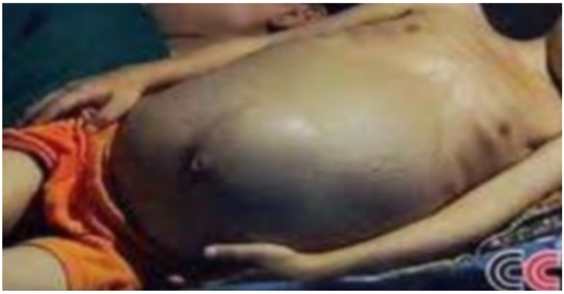
DISORDER	GENOTYPIC ABNORMALITY	CLINICAL PHENOTYPE
β-THALASSEMIA		
Thalassemia major (Cooley anemia)	Homozygous β^0-thalassemia	Severe hemolysis, ineffective erythropoiesis, transfusion dependency, hepatosplenomegaly, iron overload
Thalassemia intermedia	Compound heterozygous β^0- and β^+-thalassemia	Moderate hemolysis, splenomegaly, moderately severe anemia, but not transfusion dependent; main life-threatening complication is iron overload
Thalassemia minor	Heterozygous β^0- and β^+-thalassemia	Microcytosis, mild anemia
α-THALASSEMIA		
Silent carrier	α-/αα	Normal complete blood count
α-Thalassemia trait	αα/- - (α-thalassemia 1, *cis* deletion) *or* α -/α - (α-thalassemia 2, *trans* deletion)	Mild microcytic anemia
Hemoglobin H	α -/- -	Microcytic anemia and mild hemolysis; not transfusion dependent*
Hydrops fetalis	- -/- -	Severe anemia, intrauterine anasarca from congestive heart failure; death in utero or at birth

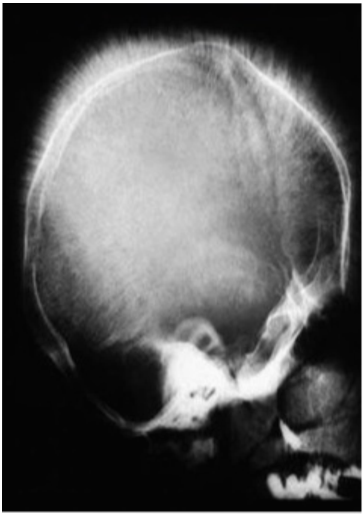

α-Thalassemia genotypes
α
α
α
α
Normal
"Classic" α-thalassemia
α-Thalassemia with Hb constant spring
α⁺- Thal trait
("Mild" trait, silent carrier)
α
α
α
Heterozygous Hb CS
(resembles α⁺-Thal)
α
αCS
α
α
~1% Hb CS
α⁰- Thal trait
("Typical" α-Thal trait)
α
α
Homozygous Hb CS
(more severe than
α⁰-Thal)
α
αCS
α
αCS
~5% Hb CS
Hemoglobin H disease
α
Hb H disease
with Hb CS
α
αCS
2%–3% Hb CS
10%–15% Hb H
Hb Bart's-hydrops fetalis
Hydrops fetalis not
observed with αCS





Thalassemic/Chipmunk facies





Hair on end appearance

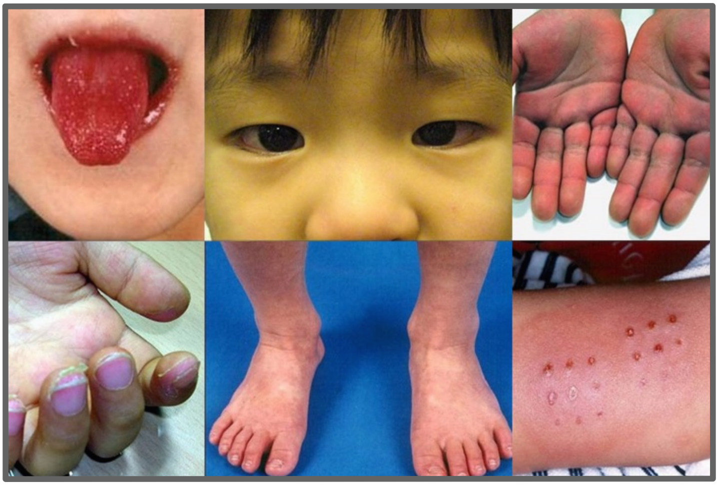
KAWASAKI DISEASE

Medium vessel vasculitis involving coronary artery

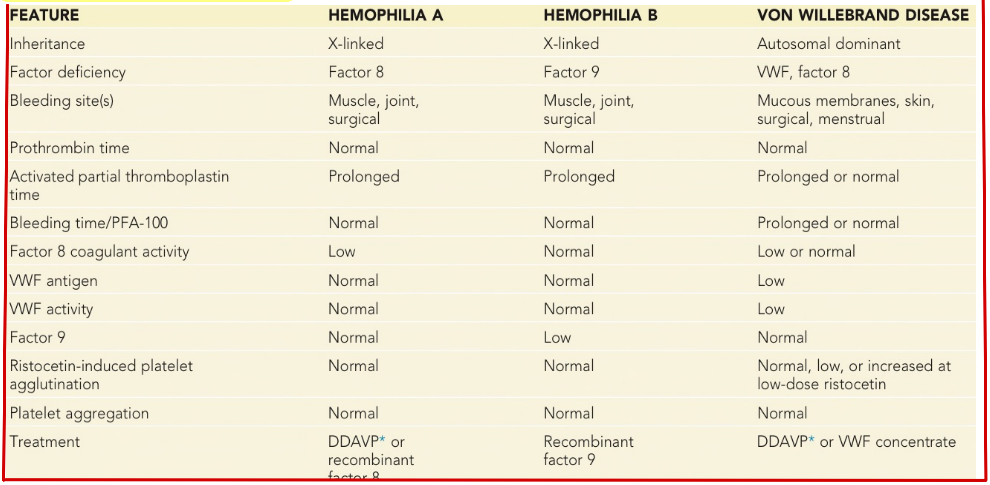
Most common vasculitis in childhood

CREAM CRITERIA

- Conjunctivitis
- Rash (Polymorphic truncal rash)
- Edema (of extremities in acute phase; skin peeling in subacute phase)
- Adenopathy (cervical lymphadenopathy; single >1.5 cm)
- Mucosal involvement (Strawberry tongue, erythematous lips)



HEMOSTATIC DISORDERS



FEATURE	HEMOPHILIA A	HEMOPHILIA B	VON WILLEBRAND DISEASE
Inheritance	X-linked	X-linked	Autosomal dominant
Factor deficiency	Factor 8	Factor 9	VWF, factor 8
Bleeding site(s)	Muscle, joint, surgical	Muscle, joint, surgical	Mucous membranes, skin, surgical, menstrual
Prothrombin time	Normal	Normal	Normal
Activated partial thromboplastin time	Prolonged	Prolonged	Prolonged or normal
Bleeding time/PFA-100	Normal	Normal	Prolonged or normal
Factor 8 coagulant activity	Low	Normal	Low or normal
VWF antigen	Normal	Normal	Low
VWF activity	Normal	Normal	Low
Factor 9	Normal	Low	Normal
Ristocetin-induced platelet agglutination	Normal	Normal	Normal, low, or increased at low-dose ristocetin
Platelet aggregation	Normal	Normal	Normal
Treatment	DDAVP* or recombinant factor 8	Recombinant factor 9	DDAVP* or VWF concentrate

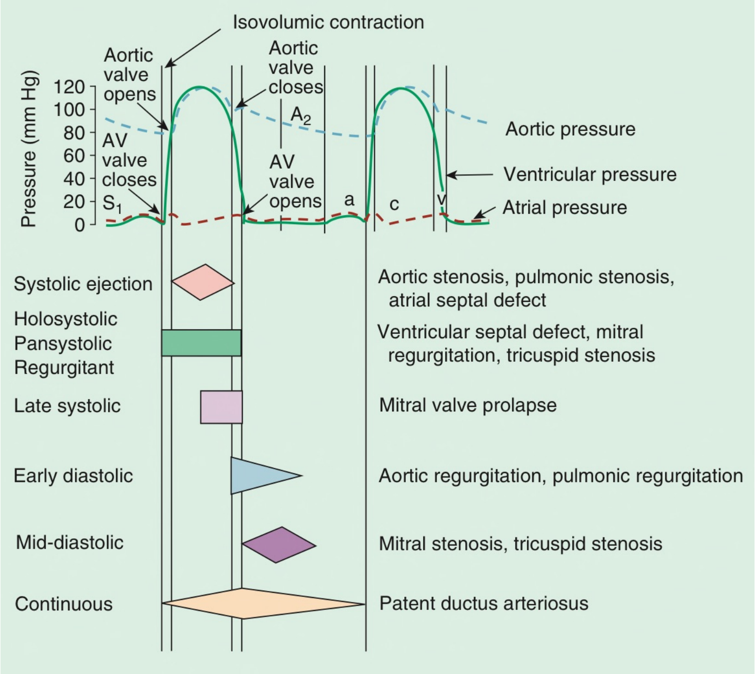
Paediatric Cardiology

SYNDROME	CARDIAC FEATURES
Trisomy 21 (Down syndrome)	Endocardial cushion defect, VSD, ASD, PDA
Trisomy 18	VSD, ASD, PDA, PS
Trisomy 13	VSD, ASD, PDA, dextrocardia
XO (Turner syndrome)	Coarctation of aorta, aortic stenosis
CHARGE association (coloboma, heart, atresia choanae, retardation, genital and ear anomalies)	TOF, aortic arch and conotruncal anomalies*
22q11 (DiGeorge) syndrome	Aortic arch anomalies, conotruncal anomalies*
VACTERL association† (vertebral, anal, cardiac, tracheoesophageal, radial, renal, limb anomalies)	VSD
Marfan syndrome	Dilated and dissecting aorta, aortic valve regurgitation, mitral valve prolapse
William syndrome	Supravalvular aortic stenosis, peripheral pulmonary stenosis
Infant of diabetic mother	Hypertrophic cardiomyopathy, VSD, conotruncal anomalies
Asplenia syndrome	Complex cyanotic heart lesions, anomalous pulmonary venous return, dextrocardia, single ventricle, single AV valve
Polysplenia syndrome	Azygos continuation of inferior vena cava, pulmonary atresia, dextrocardia, single ventricle
Fetal alcohol syndrome	VSD, ASD
Fetal hydantoin syndrome	TGA, VSD, TOF

SYSTEMIC DISEASE	CARDIAC COMPLICATIONS
Hunter-Hurler syndrome	Valvular insufficiency, heart failure, hypertension
Duchenne dystrophy	Cardiomyopathy, heart failure
Pompe disease	Short PR interval, cardiomegaly, heart failure, arrhythmias
Kawasaki disease	Coronary artery aneurysm, thrombosis, myocardial infarction, myocarditis
Marfan syndrome	Aortic and mitral insufficiency, dissecting aortic aneurysm
Juvenile rheumatoid arthritis	Pericarditis
Systemic lupus erythematosus	Pericarditis, Libman-Sacks endocarditis, congenital AV block
Lyme disease	Arrhythmias, myocarditis, heart failure
Graves disease (hyperthyroidism)	Tachycardia, arrhythmias, heart failure
Tuberous sclerosis	Cardiac rhabdomyoma
Neurofibromatosis	Pulmonic stenosis, coarctation of aorta

Reference: Essential Pediatrics Ghai (Pg 396)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 535)

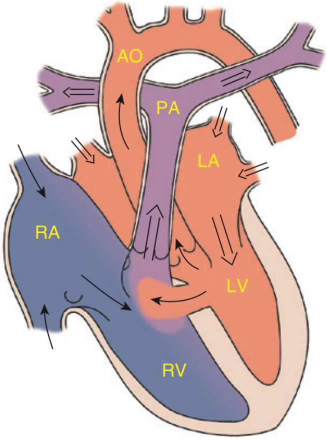
HEART MURMURS


Isovolumic contraction
Pressure (mm Hg)
120
100
80
60
40
20
0
Aortic valve opens
AV valve closes
S1
Aortic valve closes
A2
AV valve opens
a
c
v
Aortic pressure
Ventricular pressure
Atrial pressure
Systolic ejection
Aortic stenosis, pulmonic stenosis, atrial septal defect
Holosystolic Pansystolic Regurgitant
Ventricular septal defect, mitral regurgitation, tricuspid stenosis
Late systolic
Mitral valve prolapse
Early diastolic
Aortic regurgitation, pulmonic regurgitation
Mid-diastolic
Mitral stenosis, tricuspid stenosis
Continuous
Patent ductus arteriosus

	SHUNTING		
STENOTIC	**RIGHT → LEFT**	**LEFT → RIGHT**	**MIXING**
Aortic stenosis	Tetralogy	Patent ductus arteriosus	Truncus
Pulmonary stenosis	Transposition	Ventricular septal defect	TAPVR
Coarctation of the aorta	Tricuspid atresia	Atrial septal defect	HLH

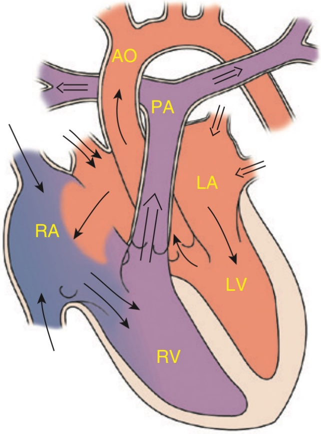
ACYANOTIC CONGENITAL HEART DISEASE

Acyanotic congenital heart disease includes left-to-right shunts resulting in an increase in pulmonary blood flow (patent ductus arteriosus [PDA], ventricular septal defect [VSD], atrial septal defect [ASD]) and obstructive lesions (aortic stenosis, pulmonary stenosis, coarctation of the aorta), which usually have normal pulmonary blood flow.



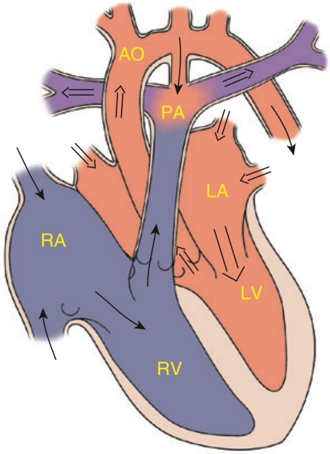
Ventricular septal defect

The typical physical finding with a VSD is a pansystolic murmur, usually heard best at the lower left sternal border.



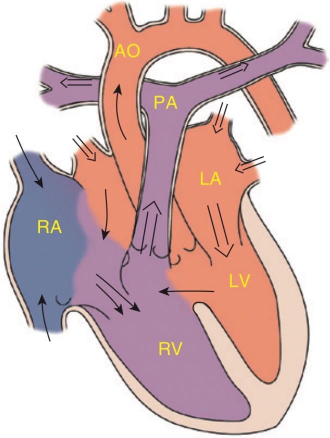
Atrial septal defect

A soft systolic ejection murmur in the region of the right ventricular outflow tract and a fixed split S2.



Patent ductus arteriosus

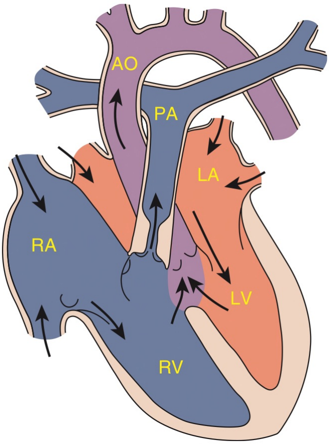
A continuous, machine-like murmur can be heard at the left infraclavicular area



Atrioventricular canal defects

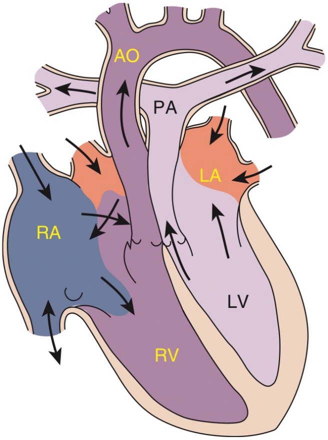
The presence of murmurs varies depending on the amount of shunting at both atrial and ventricular levels.

CYANOTIC CONGENITAL HEART DISEASE


AO
PA
LA
RA
LV
RV

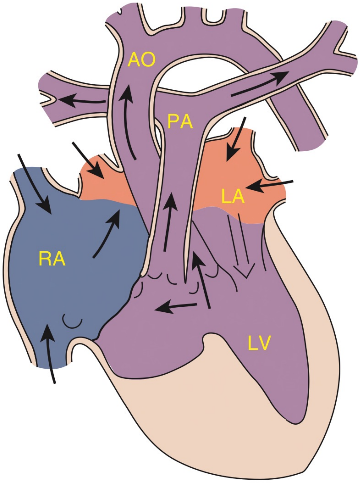
Tetralogy of Fallot

Pulmonary stenosis murmur a single S2 and right ventricular impulse at the left sternal border are typical findings.


AO
PA
LA
RA
LV
RV

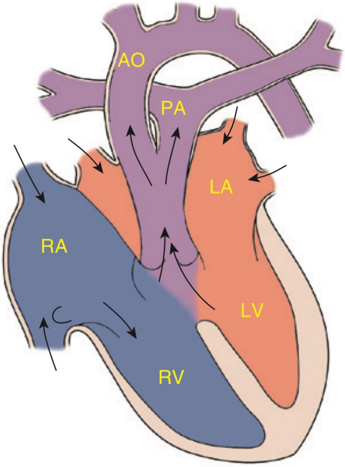
Transposition of the great vessels

A loud VSD murmur is heard. S2 is single.


AO
PA
LA
RA
LV

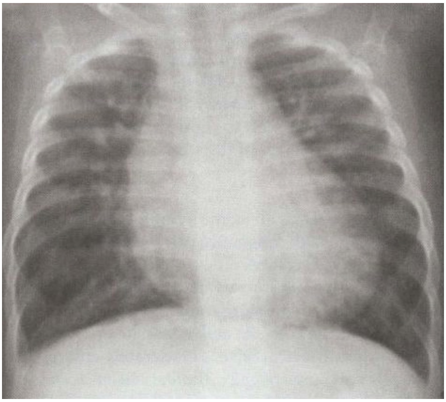
Tricuspid atresia with vsd

Infants with tricuspid atresia are usually severely cyanotic and have a single S2.


AO
PA
LA
RA
LV
RV

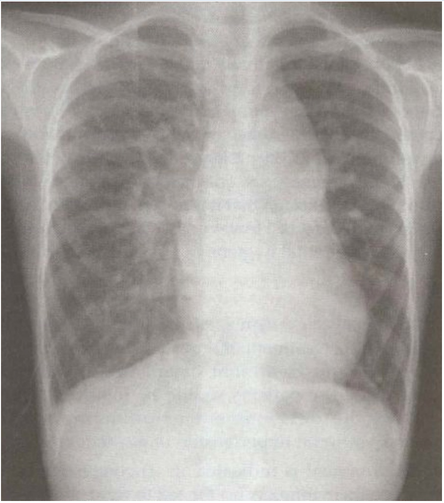
Truncus arteriosus

Systolic ejection click, and often a systolic murmur at the left sternal border.



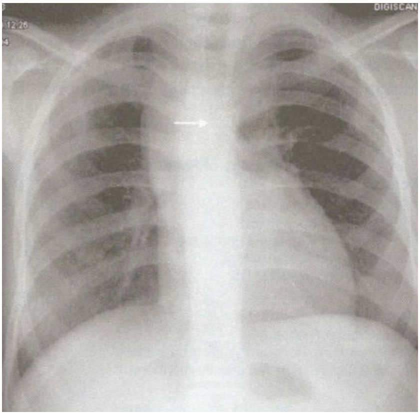
VENTRICULAR SEPTAL DEFECT.

Cardiac enlargement mainly involving the left ventricle together with increased lung vasculature as suggested by the size and increased number of end-on vessels in the lung fields



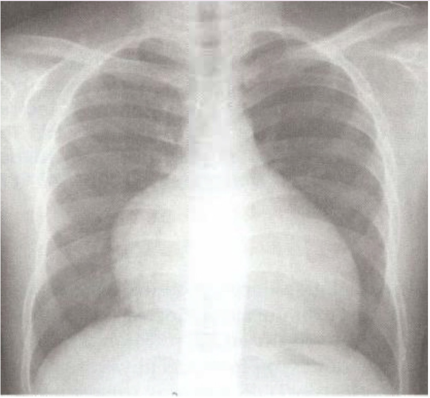
PATENT DUCTUS ARTERIOSUS

Enlargement of the aorta with a prominent aortic knuckle, large main pulmonary artery-left pulmonary artery and increased vasculature. There is no X-ray evidence of cardiac enlargement

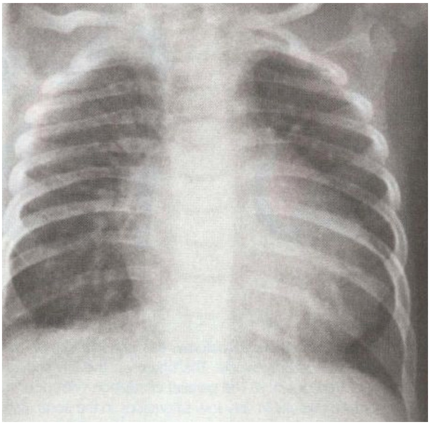


TETRALOGY OF FALLOT WITH RIGHT AORTIC ARCH.

Reduced lung vasculature as suggested by the dark lung fields, normal heart size, concavity in the region of the main pulmonary artery (pulmonarybay).

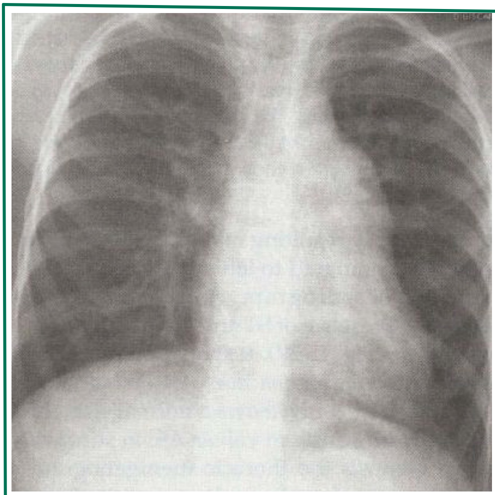


CHEST X-RAY IN EBSTEIN ANOMALY. There is considerable enlargement of the right atrium. The lung vascularity is reduced



EGG ON SIDE APPEARANCE IN TRANSPOSITION.

This characteristic appearance is seen only in about one-third cases and results from a narrow pedicle of the heart because of malpostion of great vessels

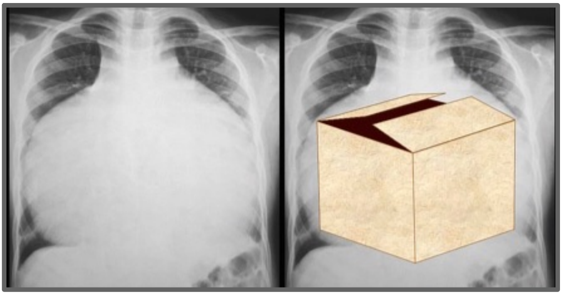


EISENMENGER SYNDROME FOLLOWING VENTRICULAR SEPTAL DEFECT.

The proximal right pulmonary artery is enlarged. There is a relative paucity of vasculature in the periphery with a sudden tapering of caliber of the right pulmonary artery (pruning)

EPSTEIN'S ANOMALY

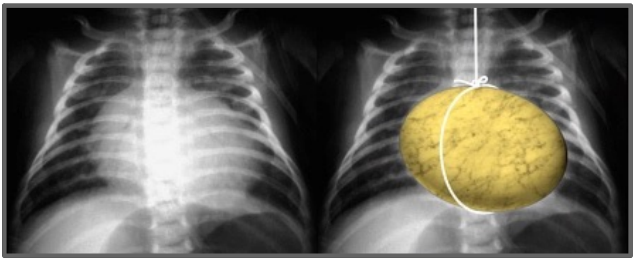
- Tricuspid valve prolapsed downwards
- Dilated right atrium – box shaped heart



TOTAL ANOMALOUS PULMONARY VENOUS CONNECTION

Anomalous connection of pulmonary vein which drains into right atrium

- **SUPRACARDIAC TAPVC**
  - Figure of 8 appearance / snowman's appearance/ cottage loaf appearance (Dilated SVC)
- **CARDIAC TAPVC** – no SVC dilatation
- **INFRACARDIAC TAPVC**- obstructive TAPVC
  (lung Congestion-> ground glass appearance)



Egg on string appearance

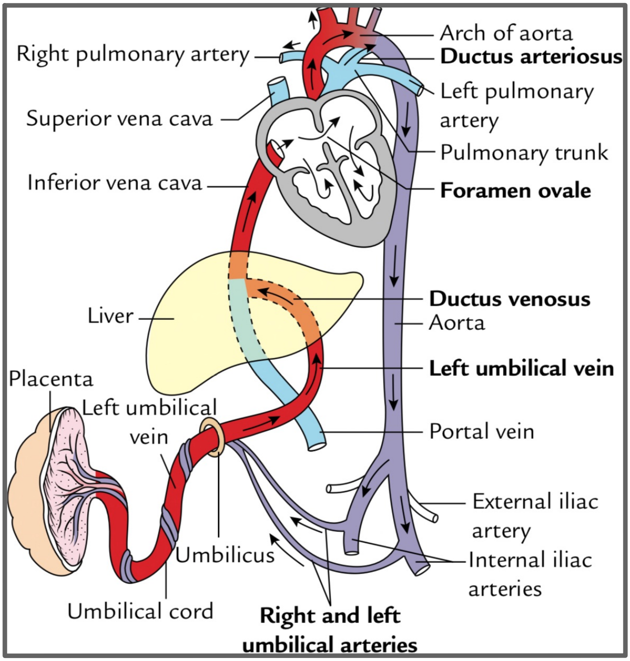
MAJOR JONES CRITERIA FOR DIAGNOSIS OF ACUTE RHEUMATIC FEVER

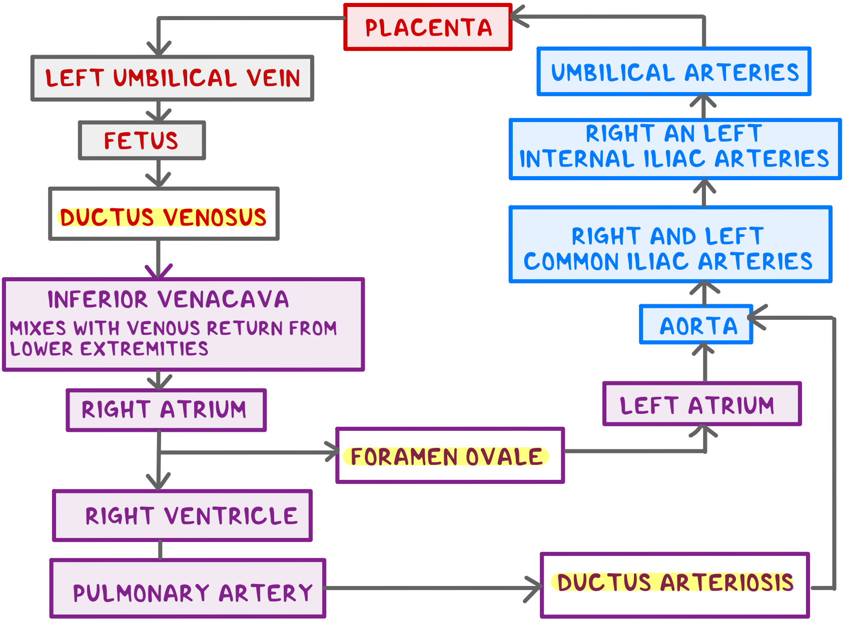
SIGN	COMMENTS
Polyarthritis	Common; swelling, limited motion, tender, erythema Migratory; involves large joints but rarely small or unusual joints, such as vertebrae
Carditis	Common; pancarditis, valves, pericardium, myocardium Tachycardia greater than explained by fever; new murmur of mitral or aortic insufficiency; Carey-Coombs mid-diastolic murmur; heart failure
Chorea (Sydenham disease)	Uncommon; manifests long after infection has resolved; more common in females; antineuronal antibody positive
Erythema marginatum	Uncommon; pink macules on trunk and proximal extremities, evolving to serpiginous border with central clearing; evanescent, elicited by application of local heat; nonpruritic
Subcutaneous nodules	Uncommon; associated with repeated episodes and severe carditis; located over extensor surface of elbows, knees, knuckles, and ankles, or scalp and spine; firm, nontender

CARDIOMYOPATHIES

FEATURE	DILATED	HYPERTROPHIC	RESTRICTIVE
Etiology	Infectious Metabolic Toxic Idiopathic	Sporadic Inherited (autosomal dominant)	Infiltrative (amyloidosis, sarcoidosis) Noninfiltrative (idiopathic, familial) Storage disease (hemochromatosis, Fabry disease) Endomyocardial disease
Hemodynamics	Decreased systolic function	Diastolic dysfunction (impaired ventricular filling)	Diastolic dysfunction (impaired ventricular filling)
Treatment	Positive inotropes Diuretics Afterload reduction β-Blockers Antiarrhythmics Anticoagulants Cardiac transplantation	β-Blockers Calcium channel blockers	Diuretics Anticoagulants Cardiac transplantation

FETAL CIRCULATION


Right pulmonary artery
Superior vena cava
Inferior vena cava
Liver
Placenta
Left umbilical vein
Umbilicus
Umbilical cord
Arch of aorta
Ductus arteriosus
Left pulmonary artery
Pulmonary trunk
Foramen ovale
Ductus venosus
Aorta
Left umbilical vein
Portal vein
External iliac artery
Internal iliac arteries
Right and left umbilical arteries



PLACENTA

LEFT UMBILICAL VEIN

FETUS

DUCTUS VENOSUS

INFERIOR VENACAVA
MIXES WITH VENOUS RETURN FROM LOWER EXTREMITIES

RIGHT ATRIUM

FORAMEN OVALE

RIGHT VENTRICLE

PULMONARY ARTERY

DUCTUS ARTERIOSIS

LEFT ATRIUM

AORTA

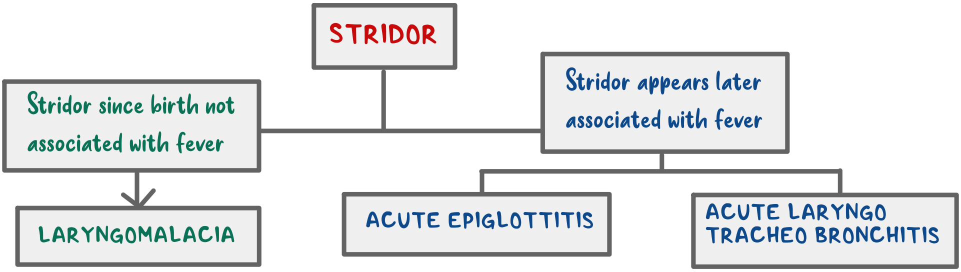
RIGHT AND LEFT COMMON ILIAC ARTERIES

RIGHT AN LEFT INTERNAL ILIAC ARTERIES

UMBILICAL ARTERIES

Respiratory Disorders

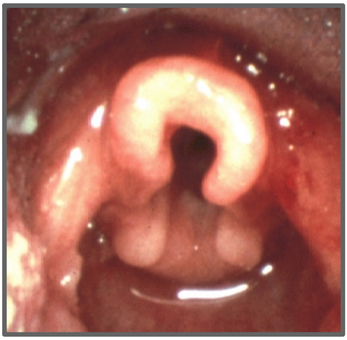
STRIDOR


STRIDOR
Stridor since birth not associated with fever
Stridor appears later associated with fever
LARYNGOMALACIA
ACUTE EPIGLOTTITIS
ACUTE LARYNGO TRACHEO BRONCHITIS

LARYNGOMALACIA

Commonest cause of strider in newborn

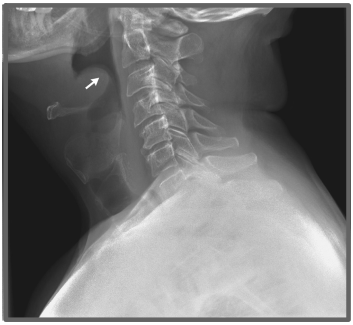
Omega shaped epiglottis



ACUTE EPIGLOTTITIS

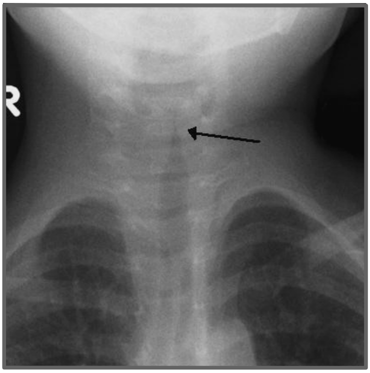
Sudden onset of stridor in foreign body

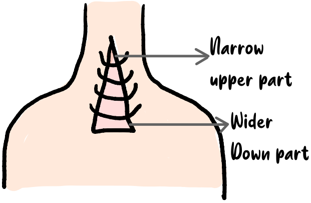
Thumb sign in X-ray (swollen epiglottis)



CROUP

ACUTE LARYNGO TRACHEO BRONCHITIS



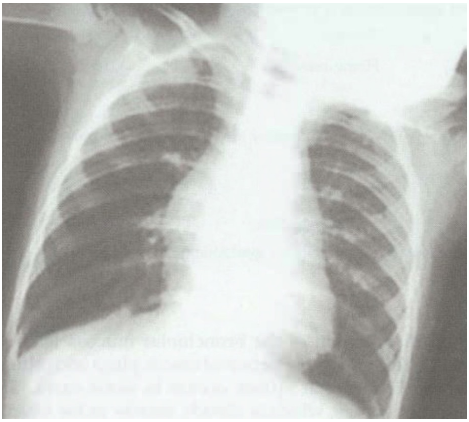

Narrow upper part
Wider Down part

STEEPLE SIGN

Reference: Essential Pediatrics Ghai (Pg371)

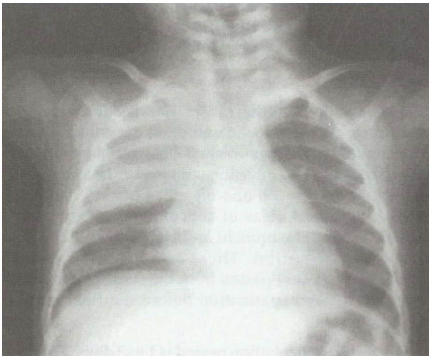
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 507)

BRONCHIOLITIS

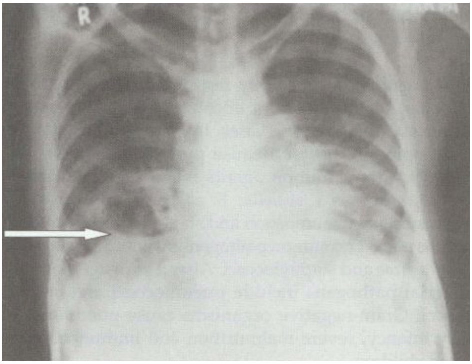


X-ray chest shows hyperinflation and infiltrates. Diaphragm is pushed down. The lung fields appear abnormally translucent.

PNEUMONIA

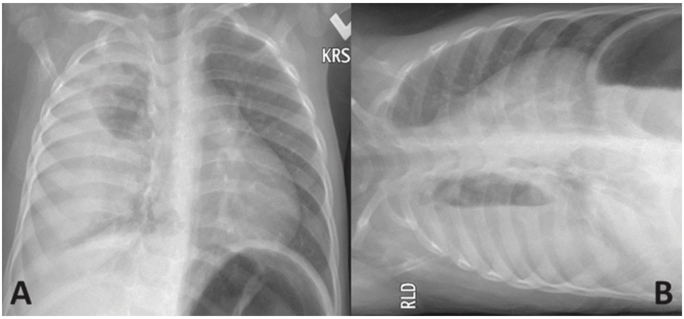


Consolidation of right upper lobe due to infection with Streptococcus pneumoniae



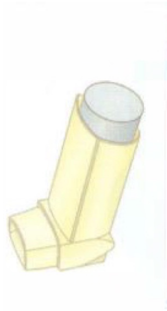
Staphylococcal pneumonia. Note multiple pneumatoceles

PARAPNEUMONIC EFFUSION.



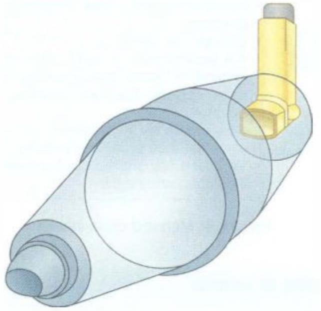
Dense opacification of the right hemithorax

INHALATION DEVICE



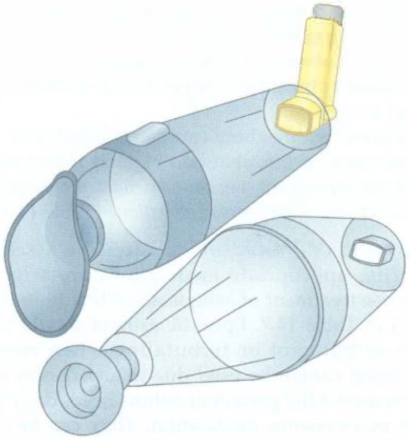
Use of metered dose inhaler

- Remove cap and shake inhaler in vertical direction
- Breathe out gently
- Put mouthpiece in mouth and at start of inspiration (which should be slow and deep), press canister down and continue to inhale deeply
- Hold breath for 10 seconds or as long as possible; then breathe out slowly
- Wait for a few seconds before repeating the inhalation once again



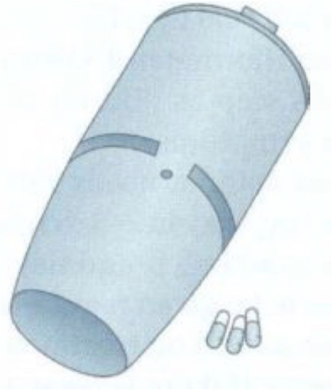
Use of metered dose inhaler

- Remove cap, shake inhaler and insert into spacer device
- Place mouthpiece of spacer in mouth
- Start breathing in and out gently and observe movements of valve
- Once breathing pattern is established, press canister and continue to breathe 5–10 times (tidal breathing)
- Remove the device from mouth and wait for 30 seconds before repeating once again



Use of MDI with spacer and baby mask

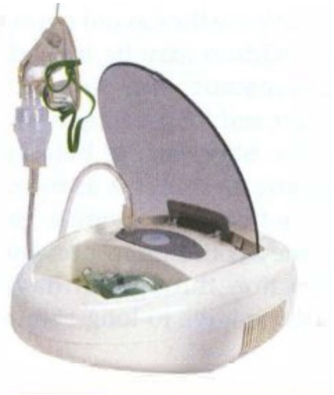
- Attach baby mask to the mouth end of spacer
- Shake MDI and insert it in the MDI end of spacer device
- Cover baby's mouth and nose with baby mask
- Press canister and encourage the child to take tidal breathing with mouth open (if possible) 5–10 times
- Remove baby mask and wait for 30–60 seconds before repeating the above step



Use of rotahaler

- Hold rotahaler vertically and insert capsule (clear end first) into square hole, make sure that top of the capsule is level with top of hole
- Hold rotahaler horizontally, twist barrel in clockwise and anticlockwise direction, this will split the capsule into two
- Breathe out gently and put mouth end of rotahaler in mouth and take deep inspiration
- Remove rotahaler from mouth and hold breathe for 10 seconds

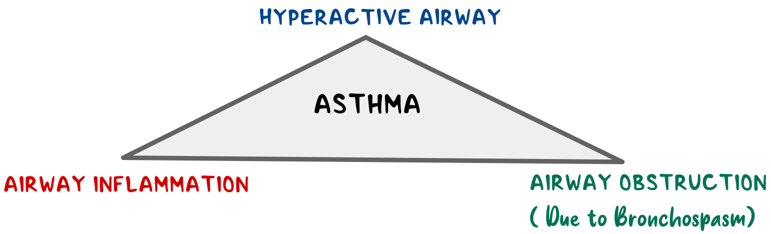
Q. Identify the inhalation device?



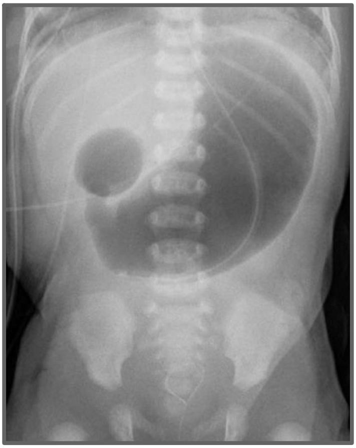
Use of nebulizer

- Connect compressor to mains
- Connect output of compressor to nebulizer chamber by the tubings provided
- Put measured amount of drug in the nebulizer chamber and add normal saline to a total of 2.5–3 m
- Switch on the compressor and look for aerosol coming out from other end of the chamber
- Attach face mask to this end of nebulizer chamber and fit it to cover nose and mouth of the child
- Encourage child to take tidal breathing with open mouth

TYPE - I HYPERSENSITIVITY REACTION (IGE MEDIATED)

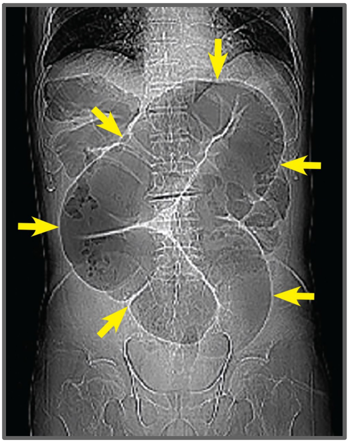


DUODENAL ATRESIA



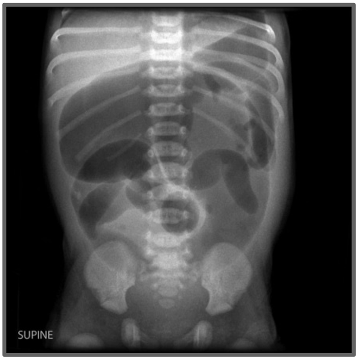
DOUBLE BUBBLE SIGN

SIGMOID VOLVULUS



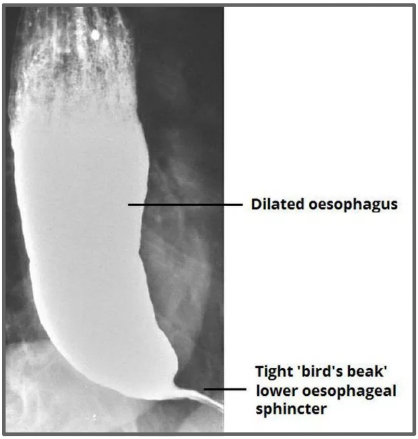
COFFEE BEAN SIGN

JEJUNUM AND ILEAL ATRESIA



MULTIPLE AIR FLUID LEVEL

ACHLASIA CARDIA

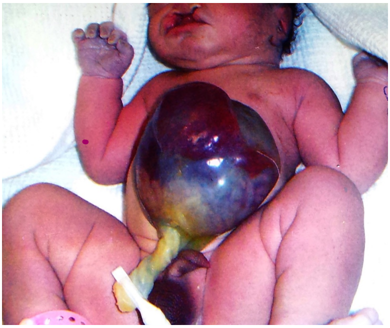

Dilated oesophagus
Tight 'bird's beak' lower oesophageal sphincter

BIRD BEAK SIGN

Reference: Essential Pediatrics Ghai (Pg 278)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 467)

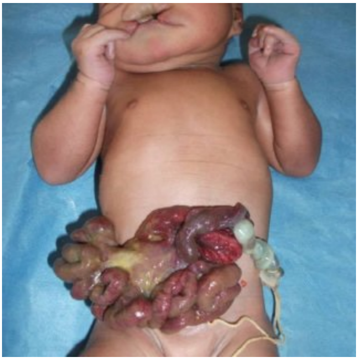
DUODENAL ATRESIA	DOUBLE BUBBLE SIGN
JEJUNUM AND ILEAL ATRESIA	MULTIPLE AIR FLUID LEVEL
SIGMOID VOLVULUS	COFFEE BEAN SIGN
ACHLASIA CARDIA	BIRD BEAK SIGN

OMPHALOCELE



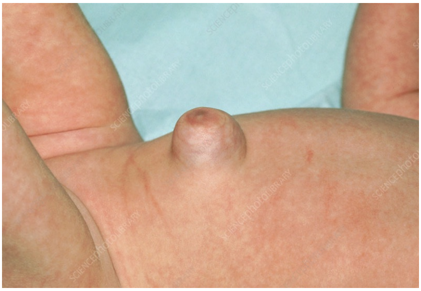
Failure of the small intestine to return into abdominal cavity from their physiological herniation into extraembryonic celom during sixth to tenth week of IUL.

GASTROSCHISIS



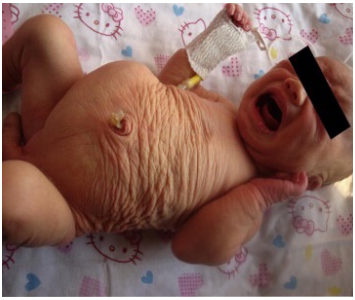
Linear defect in anterior abdominal wall through which abdominal contents herniate out

CONGENITAL UMBILICAL HERNIA



Herniation of abdominal viscera through the weak umbilical opening (poorly closed umbilicus)

PRUNE BELLY SYNDROME :



EAGLE BARRETT SYNDROME

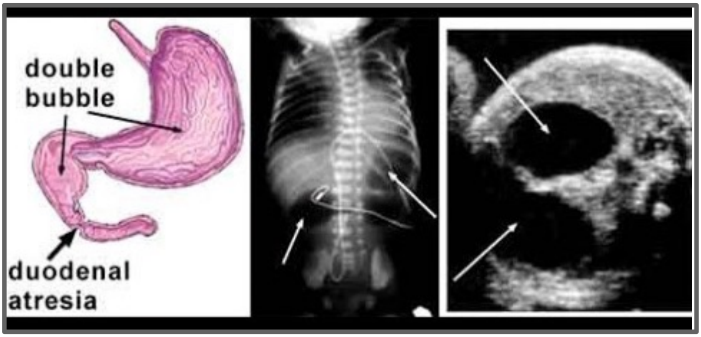
Abdominal wall defect,
Distended bladder,
Cryptochidism

DUODENAL ATRESIA

FAILURE OF RECOMILATION OF PROXIMAL GUT B/W 4 - 7 WEEK OF GESTATION

ABDOMINAL X - RAY

DOUBLE BUBBLE APPEARANCE

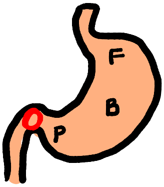

double bubble
duodenal atresia

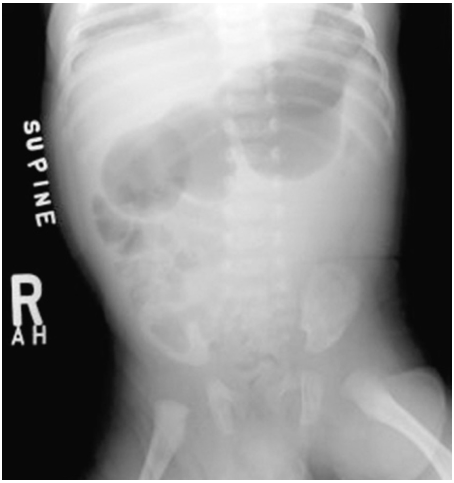
CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

PROGRESSIVE HYPERTROPHY OF CIRCULAR SMOOTH MUSCLES OF PYLORUS

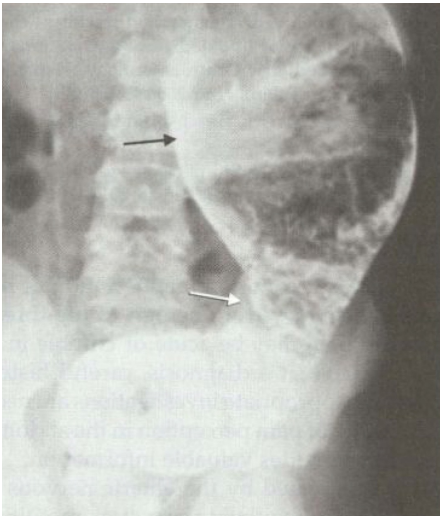
↓

GASTRIC OUTLET OBSTRUCTION





Huge, gas-filled stomach extending across the midline, with minimal air in the intestine down- stream.



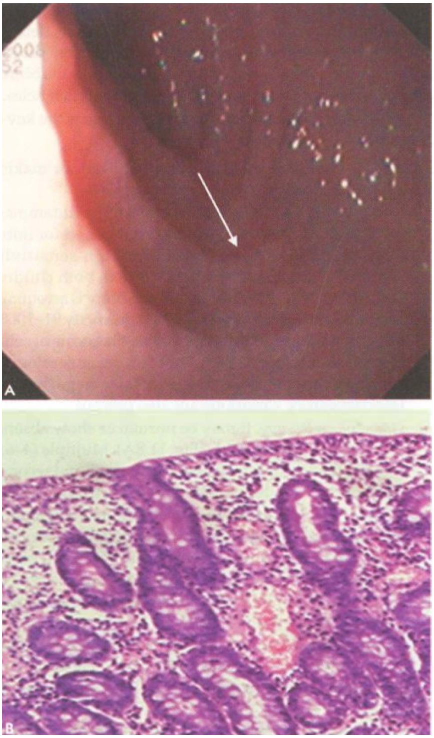
HIRSCHSPRUNG DISEASE

Barium enema showing Hirschsprung disease. Black arrow shows the dilated proximal segment and white arrow shows the transition zone

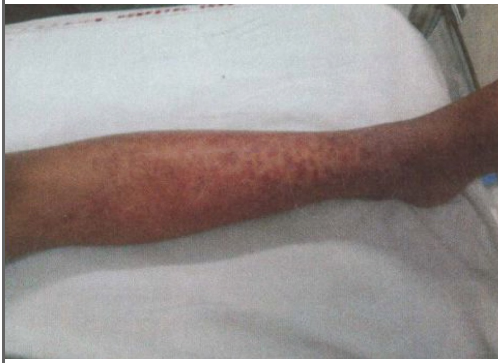
lack of ganglionic cells in the submucosal and myenteric plexuses of the distal intestine.

CELIAC DISEASE

Injury to the mucosa of the small intestine caused by the ingestion of gluten (a protein component) from wheat, rye, barley, and related grains.



(A) Upper gastrointestinal endoscopy showing scalloping of duodenal folds (arrow); (B) duodenal histology showing total villous atrophy



HENOCH-SCHONLEIN PURPURA (HSP) is the most common vasculitis in children.

Mild renal involvement indicated by microscopic hematuria and mild proteinuria is common. Serum IgA levels may be elevated.

POTTER SYNDROME

FAILURE OF URETERIC BUD TO DEVELOPE

↓

BILATERAL RENAL AGENESIS

↓

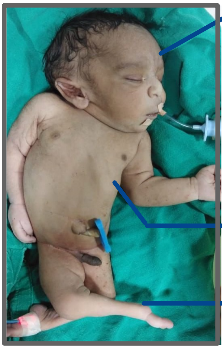
REDUCED FETAL URINE EXCRETION

↓

OLIGOHYDRAMINOS

↓

FETAL COMPRESSION



POTTER FACIES

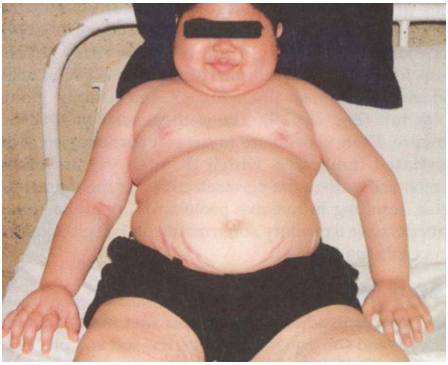
- Low set ears
- Beaked nose
- Prominent epicanthic fold
- Downward slant eyes

PULMONARY HYPOPLASIA

LIMB DEFORMITIES

NEPHROTIC SYNDROME

- **MASSIVE PROTEINURIA**
  - Urine protein excretion > 40mg /m2/ hour
  - Spot urine protein (creatinine) > 2:1
  - Urine Dipstick ⟶ 3+/4+ urine protein.
- **HYPOALBUMINEMIA**
  - Serum albumin < 2.5g/dL
- **EDEMA**
  - Periorbital puffiness - increased in the morning
- **HYPERLIPIDEMIA**
- **HYPER COAGULABILITY OF BLOOD.**



Reference: Essential Pediatrics Ghai (Pg464)

Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 617)

GLOMERULONEPHRITIS

Primary glomerulonephritis

- Acute post-streptococcal glomerulonephritis
- IgA nephropathy

Anti-glomerular basement membrane antibody disease

- Goodpasture syndrome

Glomerulonephritis of uncertain aetiology

- Minimal change nephrotic syndrome (MCNS)
- Focal segmental glomerulosclerosis (FSGS)
- Membranous nephropathy

Glomerulonephritis associated with systemic disease and infections

- Henoch-Schönlein purpura, systemic lupus erythematosus, scleroderma, polyarteritis nodosa, Wegener granulomatosis, infective endocarditis, shunt nephritis, hepatitis B and C, malaria, syphilis, toxoplasmosis, parvovirus B19, cytomegalovirus, filaria, HIV associated renal disease, Epstein-Barr virus infection.

Hereditary disorders

- Congenital nephrotic syndrome
- Familial nephritis-Alport syndrome, Sickle-cell disease

Other conditions

- Diabetes mellitus, amyloidosis, heavy metal poisoning - mercury

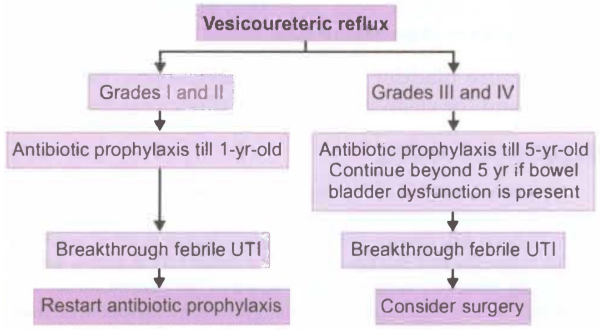
CHRONIC KIDNEY DISEASE

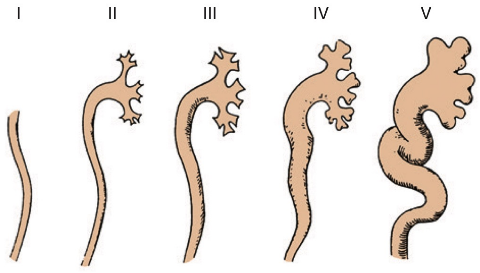
STAGE	GFR (mL/min/ 1.73 m^2)*	DESCRIPTION
1	>90	Minimal kidney damage
2	60-89	Kidney damage with mild reduction of GFR
3	30-59	Moderate reduction of GFR
4	15-29	Severe reduction of GFR
5	<15 (or dialysis)	End-stage renal disease

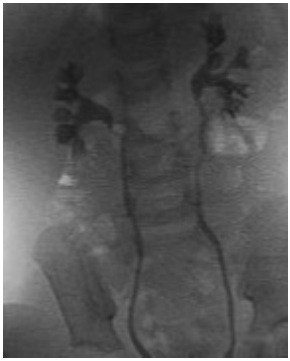
Hemodialysis in a patient with end-stage renal disease.

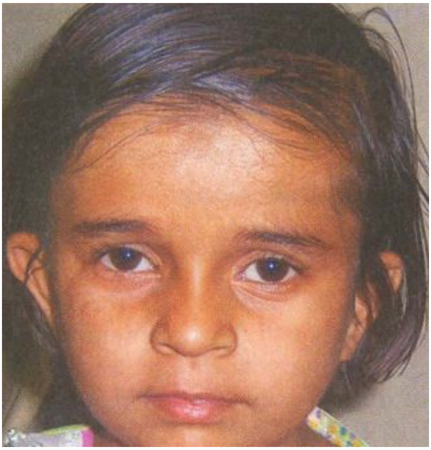
COMPLICATION	TREATMENT
1. Poor growth	Increased caloric intake, treat acidosis, treat CKD-MBD, recombinant GH
2. Anemia	Erythropoietin, iron supplementation
3. CKD-Mineral Bone Disorder/secondary hyperparathyroidism	1,25-Dihydroxyvitamin D supplementation, calcium supplementation, dietary phosphorous restriction, phosphate binders
4. Cardiovascular	
4a. Hypertension	Antihypertensive medications
4b. Left ventricular hypertrophy	Volume control
5. Electrolyte abnormalities	
5a. Hyperkalemia	Low K diet, furosemide, sodium polystyrene sulfonate
5b. Hyponatremia	Sodium supplementation
5c. Metabolic acidosis	Alkali replacement

VESICOURETERAL REFLUX

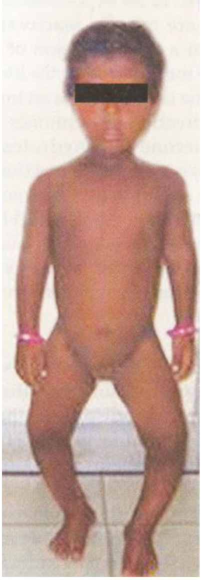

Vesicoureteric reflux
Grades I and II
Grades III and IV
Antibiotic prophylaxis till 1-yr-old
Antibiotic prophylaxis till 5-yr-old Continue beyond 5 yr if bowel bladder dysfunction is present
Breakthrough febrile UTI
Breakthrough febrile UTI
Restart antibiotic prophylaxis
Consider surgery







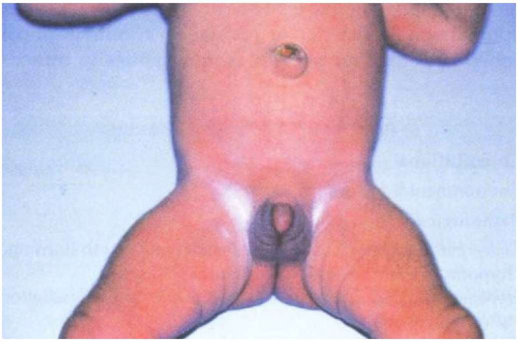
A 6-yr-old girl with short stature due to growth hormone deficiency. Note the immature facies, midfacial hypoplasia and cherubic appearance



ACHONDROPLASIA: the abnormal body proportions and facies

CONGENITAL ADRENAL HYPERPLASIA

CAH	BP	ANDEROGENIC
21 hydroxylase	Decrease	Increase
17 hydroxylase	Increase	Decrease
11 hydroxylase	Increase	Increase



Congenital adrenal hyperplasia secondary to 21- hydroxylase deficiency, Note the clitoral hypertrophy, hyper- pigmentation and increased rugosity of the labial folds giving a male appearance to the female genitalia

Reference: Essential Pediatrics Ghai (Pg 510)

Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 637)

DIAGNOSIS AND TREATMENT OF CONGENITAL ADRENAL HYPERPLASIA

Disorder	Signs And Symptoms	Laboratory Findings	Therapeutic Measures
21-Hydroxylase deficiency, nonclassic form	May be asymptomatic; precocious adrenarche, hirsutism, acne, menstrual irregularity, infertility	↑ Baseline and ACTH-stimulated 17-hydroxyprogesterone ↑ Serum androgens	Suppression with glucocorticoids
11β-Hydroxylase deficiency	Glucocorticoid deficiency	↓ Cortisol, ↑ ACTH	Glucocorticoid (hydrocortisone) replacement
		↑↑ Baseline and ACTH-stimulated 11-deoxycortisol and deoxycorticosterone	
	Ambiguous genitalia in females	↑ Serum androgens	Vaginoplasty and clitoral recession
	Postnatal virilization in males and females	↑ Serum androgens	Suppression with glucocorticoids
	Hypertension	↓ Plasma renin, hypokalemia	Suppression with glucocorticoids
3β-Hydroxysteroid dehydrogenase deficiency, classical form	Glucocorticoid deficiency	↓ Cortisol, ↑ ACTH ↑↑ Baseline and ACTH-stimulated Δ5 steroids (pregnenolone, 17-hydroxy pregnenolone, DHEA)	Glucocorticoid (hydrocortisone) replacement
	Mineralocorticoid deficiency (salt-wasting crisis)	Hyponatremia, hyperkalemia ↑ Plasma renin	Mineralocorticoid (fludrocortisone) replacement; sodium chloride supplementation
	Ambiguous genitalia in females and males	↑ DHEA, ↓ androstenedione, testosterone, and estradiol	Surgical correction of genitals and sex hormone replacement as necessary, consonant with sex of rearing
	Precocious adrenarche, disordered puberty	↑ DHEA, ↓ androstenedione, testosterone, and estradiol	Suppression with glucocorticoids
17α-Hydroxylase/17,20-lyase deficiency	Cortisol deficiency (corticosterone is an adequate glucocorticoid)	↓ Cortisol, ↑ ACTH ↑ DOC, corticosterone Low 17α-hydroxylated steroids; poor response to ACTH	Glucocorticoid (hydrocortisone) administration
	Ambiguous genitalia in males	↓ Serum androgens; poor response to hCG	Orchidopexy or removal of intra-abdominal testes; sex hormone replacement consonant with sex of rearing
	Sexual infantilism	↓ Serum androgens or estrogens	Sex hormone replacement consonant with sex of rearing
	Hypertension	↓ Plasma renin; hypokalemia	Suppression with glucocorticoids

APPROACH TO CAH

1ST DETERMINE THE TYPE OF CRISIS

SALT LOSING

SHOCK

- ♂ ♀ 21 HYDROXYLASE DEFICIENCY
- ♂ ♀ 3- OH STEROID DEFICIENCY
- ♂ ♀ ALDOSTERONE DEFICIENCY

SALT RETAINING

HYPERTENSION

- 11β-HYDROXYLASE DEFICIENCY ♂ ♀
- 17α-HYDROXYLASE DEFICIENCY ♂ ♀

2ND DETERMINE GENITALIA

MALES (XY) GENITALS

NORMAL

- 21 HYDROXYLASE DEFICIENCY
- 11β-HYDROXYLASE DEFICIENCY
- ALDOSTERONE SYNTHASE DEFICIENCY

AMBIGUOUS

- 17α-HYDROXYLASE DEFICIENCY
- 3β-HYDROXYSTEROID DEHYDROGENASE DEFICIENCY,

FEMALES (XX) GENITALS

NORMAL

- 17α-HYDROXYLASE DEFICIENCY
- ALDOSTERONE SYNTHASE DEFICIENCY

AMBIGUOUS

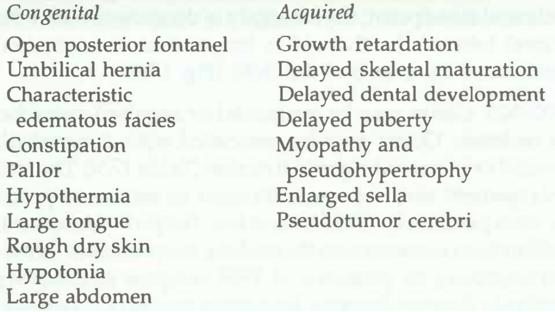
- 21 HYDROXYLASE DEFICIENCY
- 11β-HYDROXYLASE DEFICIENCY
- 3β-HYDROXYSTEROID DEHYDROGENASE DEFICIENCY,

11β-HYDROXYLASE DEFICIENCY - Most common hormonal cause of hypertension in children

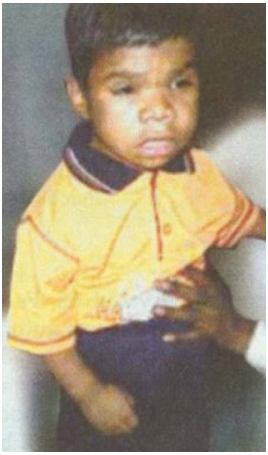
CONGENITAL HYPOTHYROIDISM

Most common cause - **THYROID DYSGENESIS (ECTOPIC THYROID)**

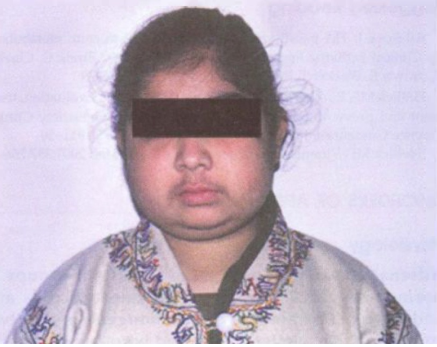
Clinical features of hypothyroidism



Congenital	*Acquired*
Open posterior fontanel	Growth retardation
Umbilical hernia	Delayed skeletal maturation
Characteristic edematous facies	Delayed dental development
Constipation	Delayed puberty
Pallor	Myopathy and pseudohypertrophy
Hypothermia	Enlarged sella
Large tongue	Pseudotumor cerebri
Rough dry skin	
Hypotonia	
Large abdomen	

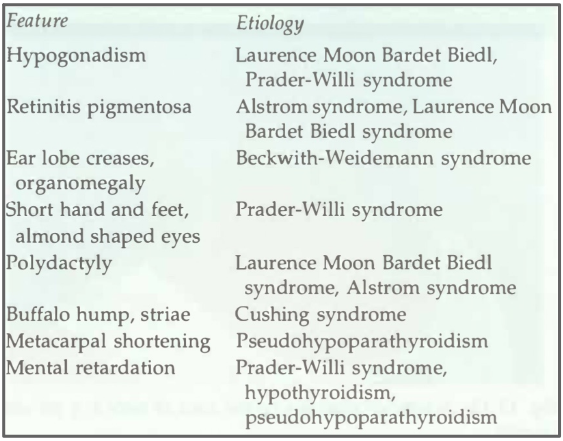


CUSHING SYNDROME



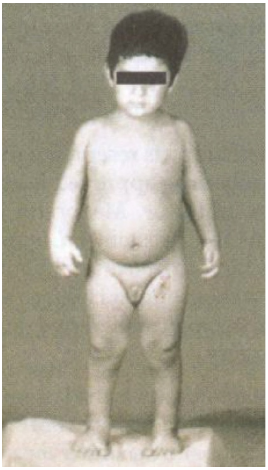
- Hypercortisolism caused by an ACTH-producing pituitary tumor.
- Classic features of Cushing syndrome such as central obesity, striae, moon facies and buffalo hump are rare in children
- Growth failure and obesity are common; other features include hypertension, hirsutism, delayed puberty, behavioral problems, bone pain and muscle weakness.

Q. Most common cause of congenital hypothyroidism?

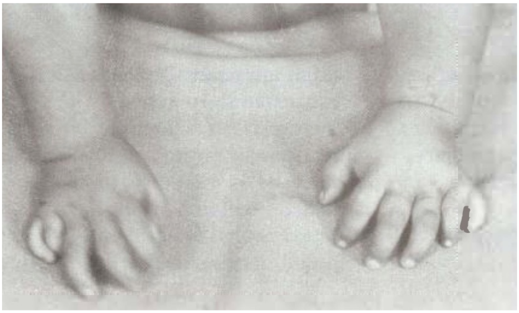


Feature	*Etiology*
Hypogonadism	Laurence Moon Bardet Biedl, Prader-Willi syndrome
Retinitis pigmentosa	Alstrom syndrome, Laurence Moon Bardet Biedl syndrome
Ear lobe creases, organomegaly	Beckwith-Weidemann syndrome
Short hand and feet, almond shaped eyes	Prader-Willi syndrome
Polydactyly	Laurence Moon Bardet Biedl syndrome, Alstrom syndrome
Buffalo hump, striae	Cushing syndrome
Metacarpal shortening	Pseudohypoparathyroidism
Mental retardation	Prader-Willi syndrome, hypothyroidism, pseudohypoparathyroidism

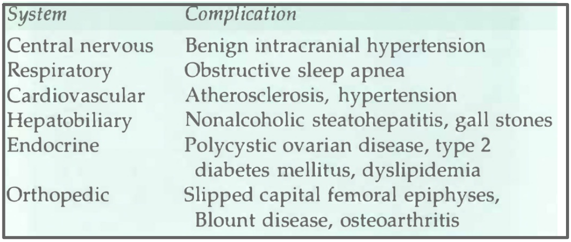
LAURENCE MOON BARDET BIEDL SYNDROME



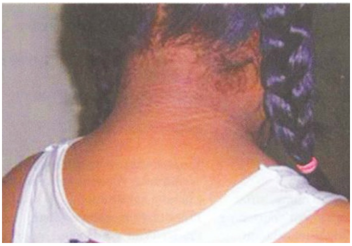
Note the central obesity and hypoplastic genitalia



Note the polydactyly



System	*Complication*
Central nervous	Benign intracranial hypertension
Respiratory	Obstructive sleep apnea
Cardiovascular	Atherosclerosis, hypertension
Hepatobiliary	Nonalcoholic steatohepatitis, gall stones
Endocrine	Polycystic ovarian disease, type 2 diabetes mellitus, dyslipidemia
Orthopedic	Slipped capital femoral epiphyses, Blount disease, osteoarthritis



Acanthosis nigricans

CAUSES OF MACROCEPHALY

- Macrocrania (increased skull thickness)
  - Achondroplasia
  - Hypochondroplasia
  - Fragile X syndrome
  - Osteopetrosis
  - Chronic, severe anemia
- Hydrocephalus (enlargement of the ventricles;
- Masses
  - Cysts
  - Arteriovenous malformations
  - Subdural fluid collections/hematoma
  - Neoplasm
- Megalencephaly (enlargement of the brain)
  - Embryological disorder causing abnormal proliferation of brain tissue
    - Neurofibromatosis
    - Tuberous sclerosis
    - Sturge-Weber syndrome
    - Sotos syndrome
    - Riley-Smith syndrome
    - Hemi-megalencephaly
  - Accumulation of abnormal metabolic substances
    - Alexander disease
    - Canavan disease
    - Gangliosidoses
    - Mucopolysaccharidoses
- Benign causes
  - Benign extracerebral collections of infancy
  - Familial macrocephaly

CAUSES OF MICROCEPHALY

PRIMARY MICROCEPHALY

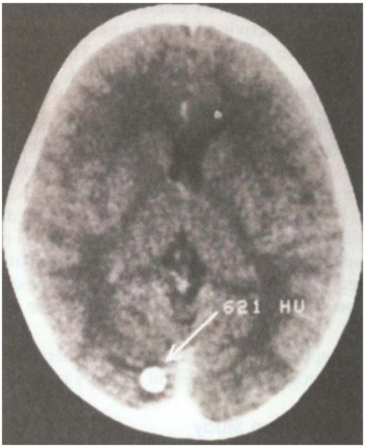
- Microcephaly vera
- Chromosomal disorders
  - Trisomy 21
  - Trisomy 13
  - Trisomy 18
  - 5p microdeletion
  - Angelman syndrome
  - Prader-Willi syndrome
- Central nervous system (CNS) malformati
  - Holoprosencephaly
  - Encephalocele
  - Hydranencephaly
- CNS migrational disorder
  - Lissencephaly
  - Schizencephaly
  - Pachygyria
  - Micropolygyria
  - Agenesis of the corpus callosum
- Sex-linked microcephaly syndromes
  - Smith-Lemli-Opitz syndrome
  - Cornelia de Lange syndrome
  - Seckel dwarfism syndrome
  - Cockayne syndrome
  - Rubinstein-Taybi syndrome
  - Hallermann-Streiff syndrome

SECONDARY (ACQUIRED) MICROCEPHALY

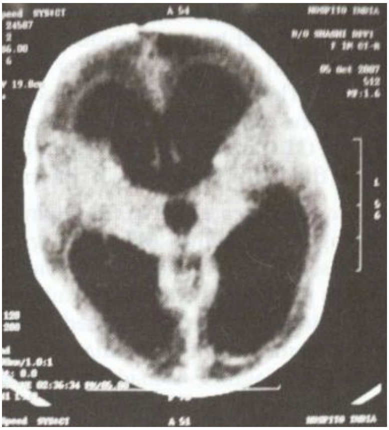
- Infections (congenital)
  - Rubella
  - Cytomegalovirus
  - Toxoplasmosis
  - Syphilis
  - Human immunodeficiency virus (HIV)
  - Zika virus
- Infections (noncongenital)
  - Meningitis
  - Encephalitis
- Stroke
- Toxic
  - Radiation exposure—fetal
  - Fetal alcohol syndrome
  - Maternal phenylketonuria
- Hypoxic-ischemic or other severe brain injury
  - Periventricular leukomalacia
- Systemic disease
  - Chronic cardiac or pulmonary disease
  - Chronic renal disease
  - Malnutrition
- Craniosynostosis totalis

Reference: Essential Pediatrics Ghai (Pg 549)

Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg681)

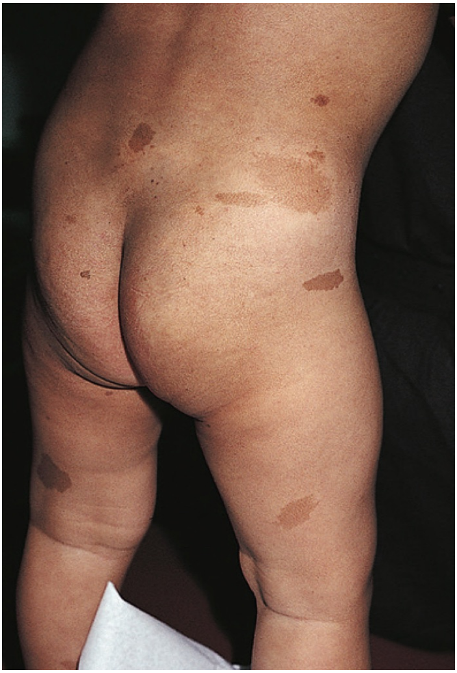


Noncontrast enhanced CT of brain showing a calcified cyst



Contrast enhanced CT of brain showing communicating hydrocephalus and periventricular ooze in a child with tubercular meningitis

NEUROFIBROMATOSIS



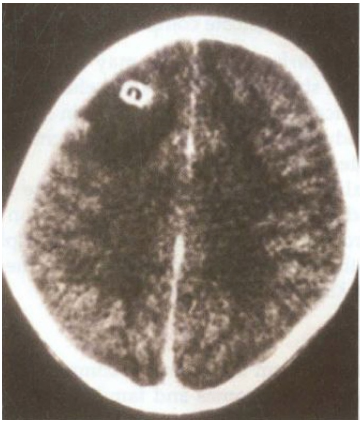
- Neurofibromatosis type 1 (NF1), also known as von Reckling- hausen disease
- Autosomal-dominant disorder
- Caused by mutations of the NF1 gene, which codes for a tumor suppressor gene, neurofibromin.
- The cardinal features of neurofibromatosis are café-au-lait spots, axillary or inguinal freckling, cutaneous neurofibromas, and iris hamartomas (Lisch nodules).

Q. Neurofibromatosis is due to mutation in?

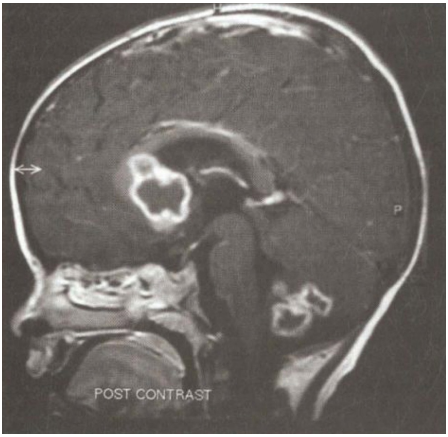
TUBEROUS SCLEROSIS COMPLEX

Autosomal dominantly neuro cutaneous disorder

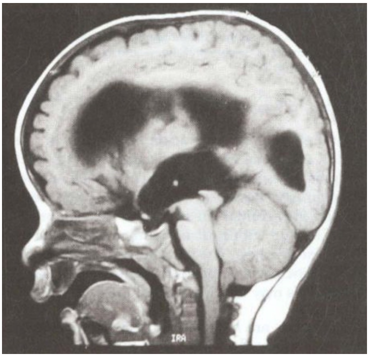
Cardinal features are skin lesions, convulsion and mentalretardation. Early skin lesions are hypopigmented, ash leaf shaped macules, red or pink papules (angiofibromas) called adenoma sebaceum on face.



Contrast enhanced CT of brain showing a degenerating ring enhancing cyst with eccentric scolex and perilesional edema in right frontal lobe

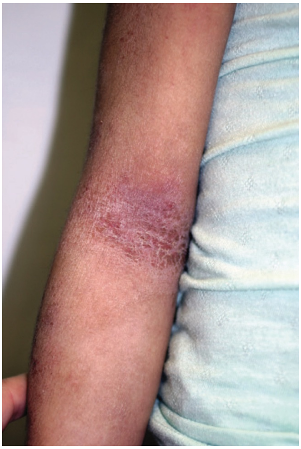


Contrast enhanced MRI (sagittal view) of brain showing enhancing irregular ring like multiple tuberculomas

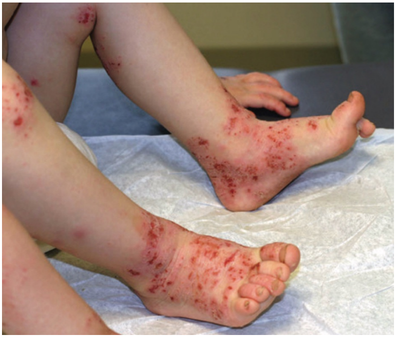


Arnold-Chiari II malformation: MRI T1W sagittal view showing obstructive hydrocephalus stretched brainstem and tonsillar herniation

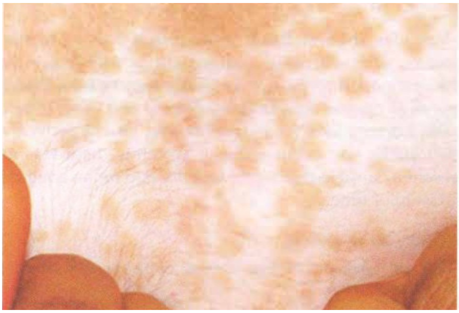
ATOPIC DERMATITIS



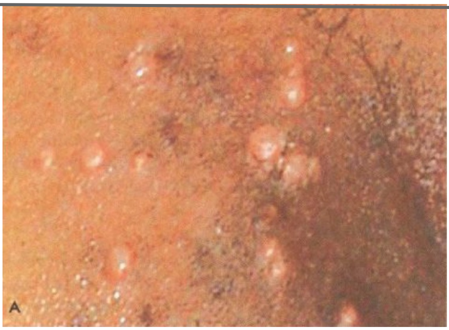
Atopic dermatitis is a chronic, relapsing skin disease characterized by xerosis, pruritus, and characteristic skin findings.



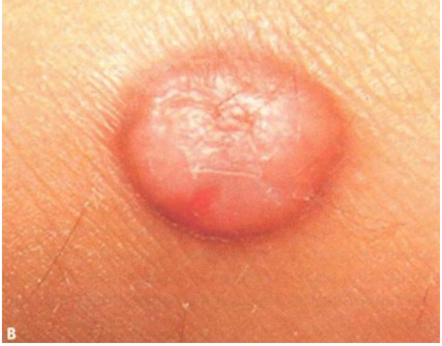
Atopic dermatitis with Staphylococcus superinfection



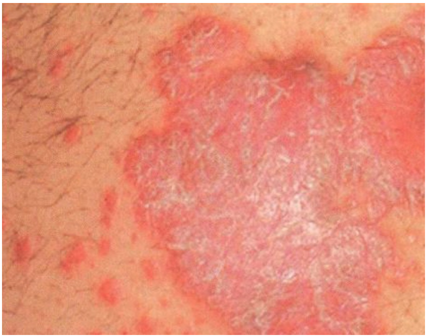
Macule, Circumscribed area of change in skin color without any change in consistency



Papule: Solid lesion, <0.5 cm;

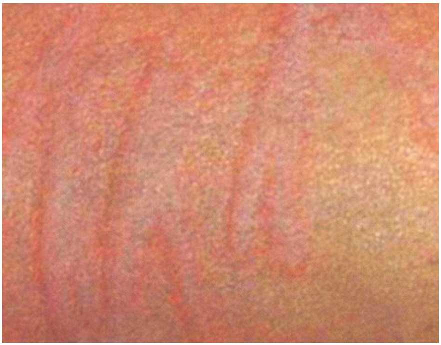


Nodule: Solid lesion, >0.5 cm

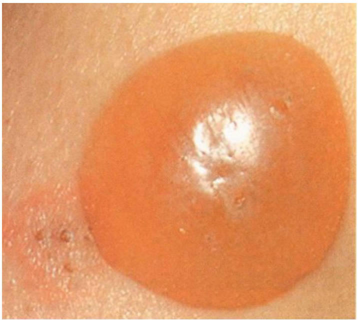


Plaque: Area of altered skin consistency with a surface area of which is greater than its depth

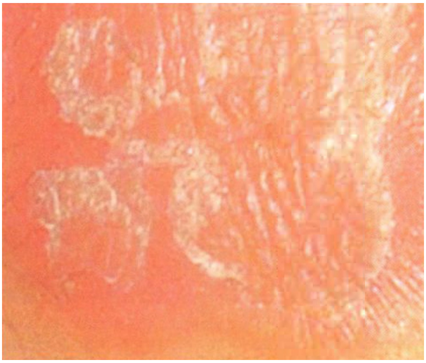
Reference: Essential Pediatrics Ghai (Pg 672)
Reference: ESSENTIALS OF PEDIATRICS, Nelson (Pg 721)



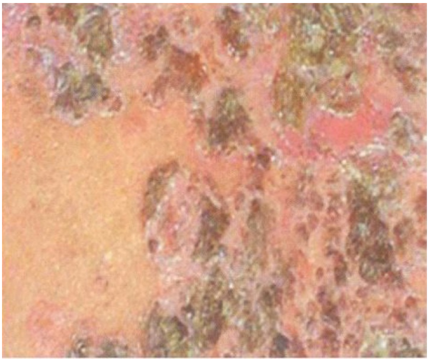
Wheal: Evanescent, pale erythematous raised lesion which disappears within 24-48 hr



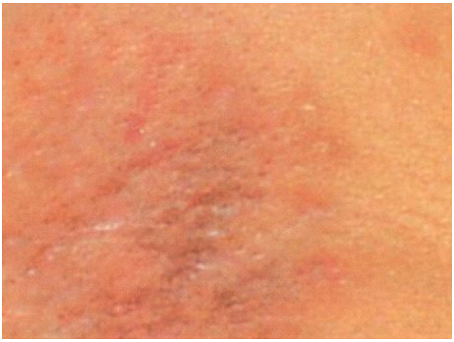
Blister: Circumscribed, elevated, superficial fluid filled lesion



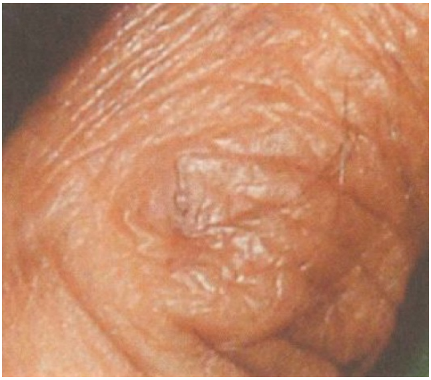
Scale: Flakes of stratum corneum



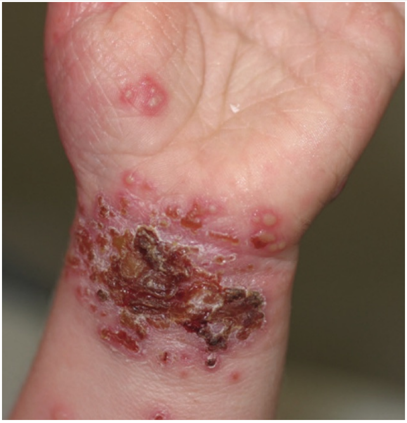
Crust: Yellow brown collection of keratin and serum



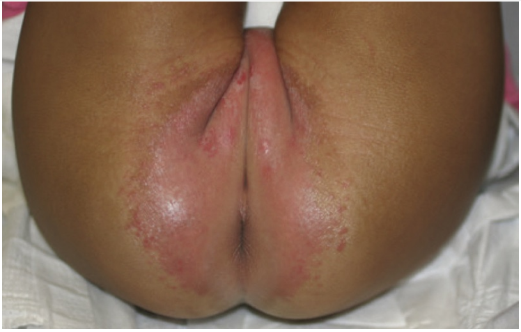
Lichenification, Thickening and hyperpigmentation of skin with increased skin markings



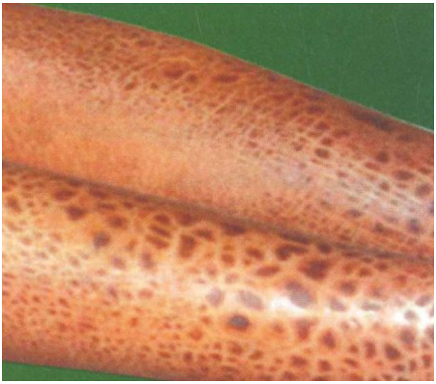
Burrow: Serpentine, thread-like, grayish curvilinear lesion, diagnostic of scabies



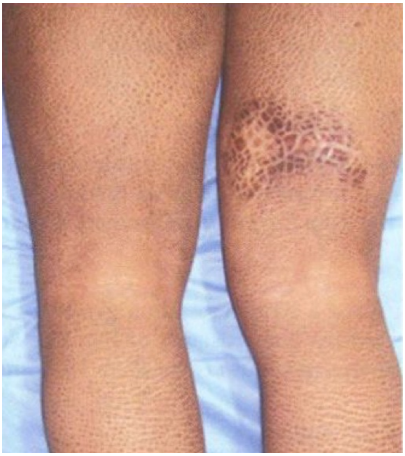
Eczema herpeticum (hand)



Irritant diaper dermatitis

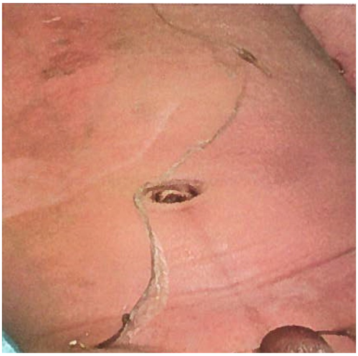


Ichthyosis vulgaris: Large scales on extremities that are attached at the center and turned up at the edge



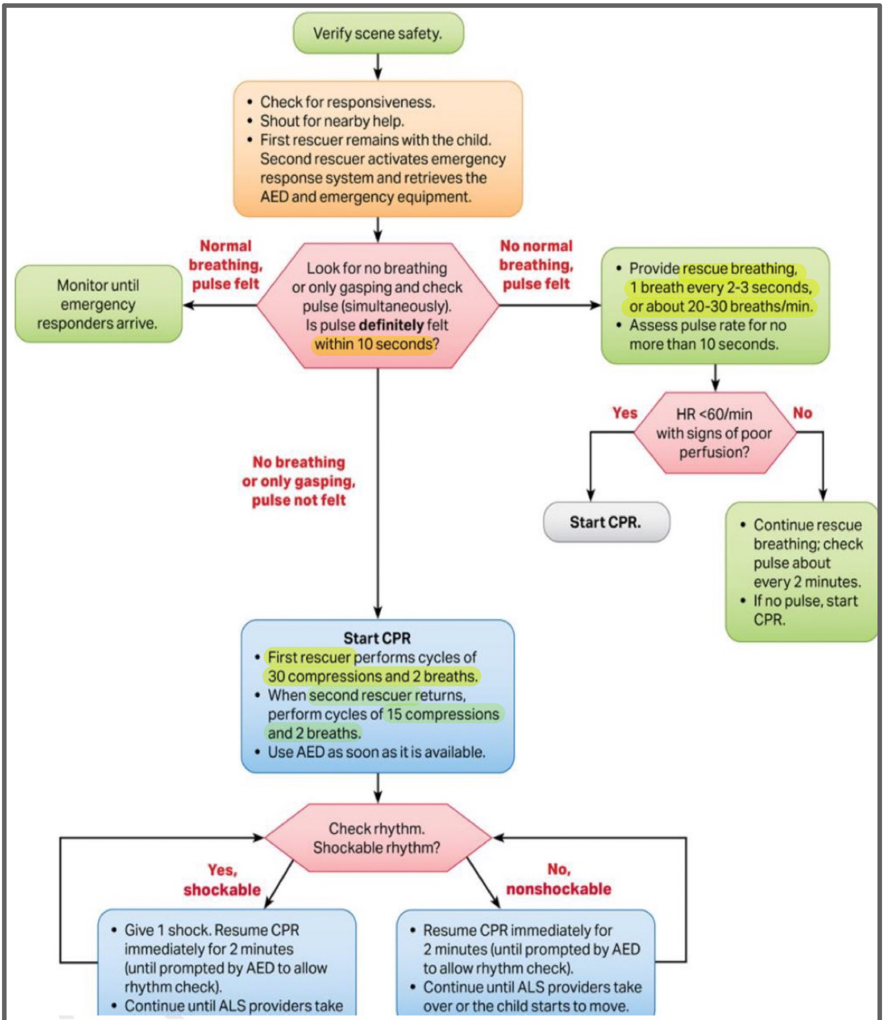
X linked ichthyosis: Large dark brown adherent scales without sparing of flexure

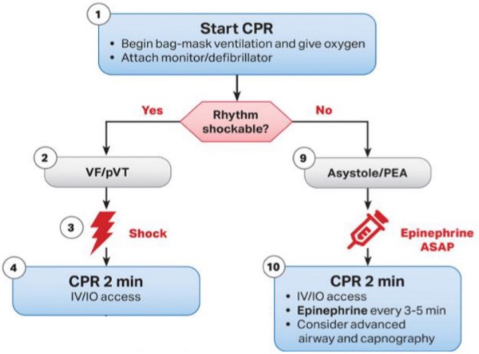
STAPHYLOCOCCAL SCALDED SKIN SYNDROME



- Mediated by hematogenously spread exotoxin produced by S. aureus present in infected site distant from the involved skin
- Erythema and tenderness is followed by superficial peeling of skin

CPR 2020 UPDATE


Verify scene safety.
• Check for responsiveness.
• Shout for nearby help.
• First rescuer remains with the child. Second rescuer activates emergency response system and retrieves the AED and emergency equipment.
Look for no breathing or only gasping and check pulse (simultaneously). Is pulse **definitely** felt within 10 seconds?
Normal breathing, pulse felt
Monitor until emergency responders arrive.
No normal breathing, pulse felt
• Provide rescue breathing, 1 breath every 2-3 seconds, or about 20-30 breaths/min.
• Assess pulse rate for no more than 10 seconds.
HR <60/min with signs of poor perfusion?
Yes
Start CPR.
No
• Continue rescue breathing; check pulse about every 2 minutes.
• If no pulse, start CPR.
No breathing or only gasping, pulse not felt
Start CPR
• First rescuer performs cycles of 30 compressions and 2 breaths.
• When second rescuer returns, perform cycles of 15 compressions and 2 breaths.
• Use AED as soon as it is available.
Check rhythm. Shockable rhythm?
Yes, shockable
• Give 1 shock. Resume CPR immediately for 2 minutes (until prompted by AED to allow rhythm check).
• Continue until ALS providers take
No, nonshockable
• Resume CPR immediately for 2 minutes (until prompted by AED to allow rhythm check).
• Continue until ALS providers take over or the child starts to move.


1
Start CPR
• Begin bag-mask ventilation and give oxygen
• Attach monitor/defibrillator
Rhythm shockable?
Yes
No
2
VF/pVT
3
Shock
4
CPR 2 min
IV/IO access
9
Asystole/PEA
Epinephrine ASAP
10
CPR 2 min
• IV/IO access
• **Epinephrine** every 3-5 min
• Consider advanced airway and capnography

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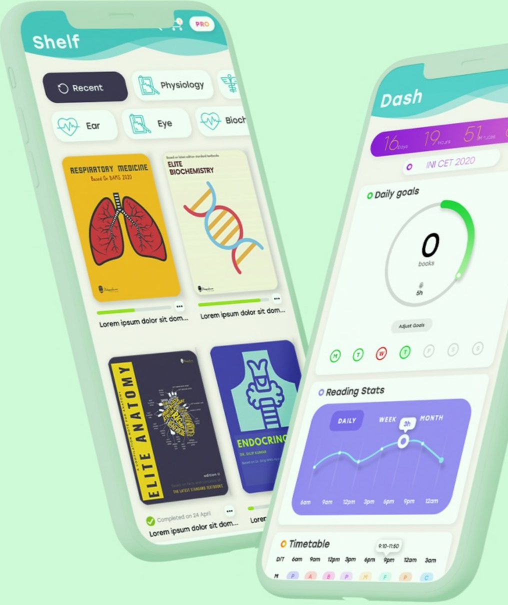
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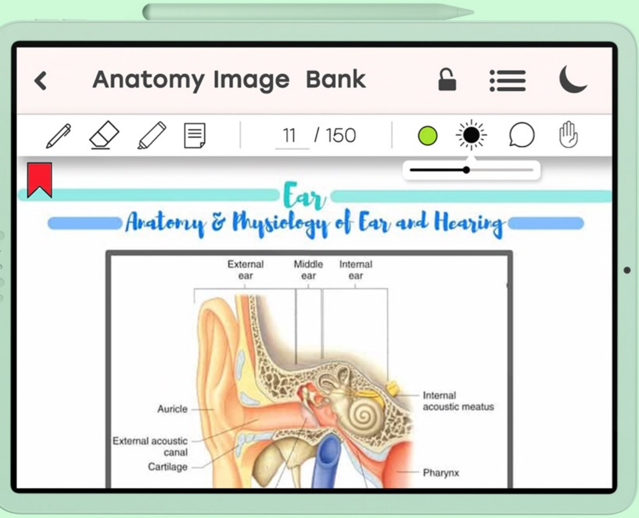
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